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Notes on the Toxicity of Tartar Emetic given Intravenously to Cattle: with Especial Reference to the Drug's Action on the Liver.

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The African trypanosomiases of cattle, due to two or more species of trypanosomes, but symptomatically so similar that the word "nagana" is used as a common name for them all, are best treated by intravenous injections of tartar emetic, either potassium antimonyl tartrate or sodium antimonyl tartrate, and thousands of such cases are so treated every year throughout Zululand, Rhodesia, Nyasaland, Tanganyika Territory, Uganda and other African countries.

It is probable that very soon better results will be achieved with some synthetic organic antimony compound as, for example, with the German preparation *Antimosan*, which is at present being tested by my colleagues in East Africa, but all such complex products are expensive, and so far none that has been fully tested on the larger domestic animals has given results sufficiently superior to those obtained with tartar emetic to justify the cost of its use in routine treatment of these animals.

Although the sodium salt is more expensive and less readily procurable than the potassium one, many people prefer to use sodium antimonyl tartrate, as this is somewhat less toxic. Khalil (1926) points out, however, that it is also less stable and may decompose on boiling to produce a highly toxic white precipitate. The trypanocidal action of both is clinically the same, so choice must be based on the difference in price, procurability and toxicity. Personally, I have always used the potassium salt.

The routine treatment of an average fly-struck native ox, weighing about 500 lb., is a series of intravenous injections of about one gramme of tartar emetic. The procedure I now adopt is to give 25 c.c. of a 4 per cent. aqueous solution, and to repeat this dose at seven-day intervals until it is hoped that cure has been effected, or until further treatment along the same lines becomes impossible owing to the development of symptoms of antimony poisoning. If no such symptoms have developed after seven or eight injections, treatment is suspended until time reveals whether cure has been brought about or not. If trypanosomes reappear in the blood, a second similar course of injections is given.

No one can deny the high trypanocidal action of tartar emetic, and that many fly-struck cattle which show what one may call a normal resistance to antimony poisoning can be cured; but the problem is how to know whether an animal has such a normal resistance before treatment is commenced, for, if it has not, then the odds are in favour of its succumbing to this poisoning before it has received what is deemed to be a curative amount of the drug.

That antimony compounds are dangerous drugs is, of course, well known, and all text-books on pathology refer to the fatty degeneration and necrosis of the liver which can be brought about by antimony administration. Yet in current literature, as we shall see, not only is this particular form of poisoning almost ignored, but the whole subject of the dangers attending antimony therapy is treated lightly. For example, Curson (1926) published what is almost a monograph on the subject of treating fly-struck cattle with tartar emetic, and yet the section on the toxicology of the drug consists of brief references to the untoward results which some practitioners have found to follow occasionally the administration of the drug to human beings. This is probably because his own work was essentially mass treatment, which does not permit either (a) the detailed examination of individual cases which is necessary to distinguish deaths due to antimony poisoning from those due to trypanosomiasis and inter-current diseases, or (b) the knowledge whether any cure is temporary or permanent. I can readily understand this, because between 1913 and 1921 I treated under field conditions more than a thousand fly-struck cattle and equines without being convinced of the extremely toxic nature of the drug. (Hornby, 1921).

During the last five years I have treated a large number of fly-struck cattle under laboratory conditions which permitted me to make more detailed observations, and I now use with diffidence the drug which formerly I looked upon as almost innocuous. Under field conditions one gives a short series of injections: if the beast dies one is consoled by the thought that the disease was too far advanced; if the beast appears better after its course one thereafter loses touch with it, or one hears from the owner *his* story of its subsequent history with his often erroneous conclusions. Under laboratory conditions, *i.e.*, when the beast's number is not taken off the case-book until it dies and is examined *post-mortem*, or until its recovery is almost a certainty, results of treatment are not as satisfactory as they appear to be in the field.

Antimony poisoning, following the intravenous injection of tartar emetic, has been studied experimentally by comparatively few. Michiels (1920) used rabbits in his investigations. He found that the drug was removed from the blood within a few minutes of injection, to be fixed by the more solid tissues, particularly the liver. Elimination he found to be by all secretions and excretions. He considered that death following the administration of a single toxic dose was due to paralysis of the respiratory centre, or to simultaneous poisoning of both respiratory and vasomotor centres. The toxic action of smaller doses he associated chiefly with hypotension due to vaso-dilatation.

More recently Chopra (1927) has studied the immediate results of the injection of antimony compounds into cats. He found that a fall in the systemic blood pressure is produced, owing to lessened output by the left ventricle and to dilatation of splanchnic vessels. This dilatation causes a distinct increase in the size of the liver and spleen.

The kidneys, on the other hand, contract slightly. There is a rise of pressure in the pulmonary arteries and the *venae cavae*, accompanied by marked bronchiole constriction. Respiration is affected, and becomes shallow, irregular, jerky and thoracic. With large doses both heart and respiration may stop simultaneously. Only incidentally does he remark that symptoms of liver disturbance frequently occur after injections of antimony compounds, and states that in a majority of the cases of human beings who have taken a course of injections a mild type of jaundice supervenes.

The drug's toxic action on the liver was evident to NISHI (1923) who carried out an experimental study on the treatment of schistosomiasis with tartar emetic, and who described symptoms and lesions produced in rabbits by overdoses of the drug. The result of giving 0.02 gm. of tartar emetic per kilo body weight was restlessness, stretching, staring coat, nausea, sometimes vomiting, anorexia, diarrhœa, and death within twenty-four hours. *Post-mortem* he found degenerative changes in liver, kidneys and lymphatic glands. His description of the microscopical picture of a liver section envisages venous congestion, hæmorrhagic foci, vacuolation of parenchyma cells; all indicating a rapid degeneration which had already proceeded as far as necrosis of part or the whole of many lobules.

BRAHMACHARI and SEN (1924) point out that the chief route of excretion of antimony is by the urine, and that the rate of excretion goes up with the amount given. Thus although after the first two or three injections at three or four-day intervals there is a tendency towards accumulation of the metal in the tissues, and particularly in the liver, yet when the concentration in this organ reaches a certain level, which they call the Maximum Concentration Limit, then the rate of excretion becomes much higher to prevent this limit being passed. One wonders whether this limit is lowest, and so most easily and disastrously passed, in the livers of those animals which are most susceptible to the hepatic form of antimony poisoning.

From experimental evidence alone, then, it can be seen that the toxic effects of antimony are varied in character. Circulatory changes would account for mild symptoms of dyspnœa, cyanosis, etc., while marked respiratory disturbances can be associated with extreme changes in the pulmonary pressure and contraction of bronchi. (CHOPRA, *loc. cit.*). A further set of symptoms, including diarrhœa, albuminuria, metallic taste, nausea and vomiting, might be produced by the irritation of the drug's passage through the excretory organs. Finally, there is the syndrome which is produced when the concentration of the drug in the liver passes the Maximum (safe) Concentration Limit and causes rapid necrosis of the parenchyma.

It has recently been suggested that some, if not much, of the toxic action of tartar emetic is due to impurities. GRIFFITH-JONES (1926) pointed out that commercial tartar emetic almost invariably contains lead and arsenic. KHALIL (1926) refers to these analyses and states that tartar emetic free from lead is appreciably less toxic than impure samples. I carried out some experiments to find out if the presence of lead as an impurity in tartar emetic enhanced the drug's toxic action on the liver, but I was unable to detect any such effect. (HORNBY, 1927.) It has been often brought to my notice, however that different samples of commercial tartar emetic appear to differ in their liability to cause fainting in cattle immediately after injection, and this difference, if real, may be one of freedom from impurities.

Although not used as much now as in recent former years, yet tartar emetic is still used extensively in human medicine for the treatment of schistosomiases, Leishmaniases, filariases and leprosy. I was surprised, then, that among the recent literature of antimony therapy I could find so few references to the drug's action toxic on the liver. For instance, in the monograph on schistosomiasis japonica by FAUST and MELENEY (1924), with its many references to previous publications, symptoms of antimony poisoning are stated to be cough, nausea, dizziness, weakness, vomiting, and later, epigastric distress, rise of temperature and diarrhœa. Of these, only epigastric distress can be connected with liver breakdown, and this association is not made by the authors.

Similarly, in the monograph on kala azar by NAPIER and MUIR (1923), the complications attributable to antimony injections are stated to be coughing, vomiting, joint and muscular pains, eruptions, and slowing of the heart. Here again the possible connection between joint and muscular pains and liver derangement is not emphasised in the text.

According to RODRIGUEZ and EUBANOS (1923), intravenous injections of tartar emetic given to lepers are particularly liable to cause nephritis. This effect recalls the definite affinity for, and therefore toxic action on, the convoluted tubule cells which all tartrates possess.

The only detailed reference to liver degeneration resulting from antimony therapy which I have been able to find in human medical literature is in an article by JOHNS (1924); and he prefaces his remarks with the statement that "the entire literature to date does not record a single case of granuloma with a fatality attributed to tartar emetic." He then goes on to refer to two patients treated by him for *granuloma inguinale* who died from such poisoning. They had received doses of 18 and 20 c.c. respectively of a 1 per cent. solution that had been stepped up from 4 c.c. by an increase of 2 c.c. per injection. *Post-mortem* he noted multiple punctate hæmorrhages, absence of blood-clotting, and *acute yellow atrophy* of the liver.

In veterinary literature the best account that I can find of tartar emetic poisoning of a domestic animal is by LECKIE (1925). He describes the train of adverse symptoms in camels affected with surra which have received overdoses of tartar emetic as: cessation of rumination; going off feed; orange-coloured or blood-tinged urine; congestion of visible mucous membranes, which eventually may become yellow or orange; watering of the eyes and nose, occasionally with ulceration of the mucous membrane of the nasal cavity; a more or less generalised dermatitis; constipation; paraplegia, and muscle tremors; hæmorrhages and disorganisation of the internal eye. He attributes these symptoms primarily to the action of the antimony salt on the liver, giving rise to a fatty degeneration, so that icterus and constipation result. No doubt in his experience a certain number of camels showed transitory symptoms of circulatory and respiratory

disturbance; but for him the drug's toxic action centres around liver degeneration.

My experience with cattle is similar to Leckie's with camels. No doubt in those animals mild toxic symptoms due to rise in pulmonary pressure are set up, but overlooked. Occasionally the severer toxic symptoms of dyspnœa, cyanosis, and marked respiratory disturbance are noted, and these may be more pronounced if impure samples of the drug are used. Rarely a beast collapses and dies a few minutes or an hour or two after receiving an injection. Symptoms of metallic taste and nausea produced by excretion of the drug into the mouth and stomach cannot be detected in ruminants, and only rarely have I seen diarrhœa which could be attributed to the drug's irritation of the intestine. I am unable to attribute any definite symptoms to the drug's undoubted poisonous action on the kidneys. *Post-mortem* one often finds the kidneys pale and moist, and with the tubule cells loaded with fat, and yet the urine is normal in its naked-eye appearance. Like CURSON (*loc. cit.*) however, I have found that all the toxic effects which I have already enumerated in this paragraph are not sufficiently common or intense to cause a mortality great enough seriously to mitigate the drug's value. It was not until I realised that the acute parenchymatous hepatitis, which I met *post-mortem* in animals succumbed during treatment, was really the cause of death and directly due to antimony that I assessed correctly the toxicity of tartar emetic.

My attention was first seriously called to this form of poisoning in 1925, when Capt. G. Norman Hall and I treated without success seven native cows, the survivors of an experimental herd of thirty cows and calves which had been kept for only a few weeks close to a fly-belt, where all the older animals speedily became infected with *T. congolense*, *T. vivax*, or both. All seven were young cows in poor condition, and all but one had lost their calves. Their average weight was about 360 lb. The contemplated course of treatment was a series of injections, each of 25 c.c. of a 4 per cent. solution of tartar emetic, at five-day intervals. No ill effects appeared to follow the first two injections, although my experience is that the symptoms due to respiratory and circulatory disturbance are more likely to occur after the first than after any subsequent injection. However, one cow developed symptoms of poisoning after the third injection, and was destroyed *in extremis* three days later. The next to go was destroyed *in extremis* six days after the fourth injection. The next died two days after the fifth injection, and the next was destroyed *in extremis* three days after the sixth injection. The remaining three cows were discharged for observation after receiving four or five injections, but all relapsed, in that *T. congolense* reappeared in their blood within four weeks of cessation of treatment, and were brought into hospital again for a second course of injections, this time at seven-day intervals. None survived, however; two died, one and three days respectively after their third injection, and the last animal was destroyed *in extremis* one day after the fourth injection.

In every case symptoms of poisoning developed within twenty-four hours of the releasing, or fatal, injection, but the time of death varied from one to six days after this injection. The symptoms were not very striking; merely dulness, anorexia, constipation and progressive weakness.

Post-mortem. We found the usual pathological changes associated with advanced trypanosomiasis, such as emaciation, anæmia, cachectic œdema, and hyperplasia of lymphoid tissue. In addition, though, there was present in every instance acute necrosis of the liver. In no case was this necrosis slight; in the least affected a third of the parenchyma was destroyed; in the most severely affected it was difficult to find any intact parenchyma cells, as those which were not broken down were full of fat droplets, and obviously breaking down.

There is no doubt that this liver necrosis was directly due to the drug. In some cases of untreated trypanosomiasis, as in all wasting diseases, one may find a high degree of fatty degeneration; but I have never seen this proceed to the state of a rapid necrosis. On the other hand, it is easy enough to set up this condition of necrosis by dosing a cachectic animal with toxic doses of an antimony salt—not necessarily tartar emetic. I have seen the same syndrome develop in an animal poisoned with the compound known as Sb 212.—For example, I picked out a thin ox which had not yet recovered from severe rinderpest. I tested the functional ability of its liver by giving it an intravenous injection of phenoltetrachlorphthalein and examining its serum one hour later. As no more than a trace, less than 2 per cent., of the dye was then present in the serum, I could assume that the liver was functioning fairly well. I then gave the beast 25 c.c. of a 4 per cent. solution of tartar emetic on three successive days. The day after the third injection he was prostrated; lay on his side with head stretched out; he passed a small quantity of hard mucus-covered dung, but urine trickled away almost continuously; visible mucous membranes were pale, bluish and somewhat yellow; extremities were cold; respiration was laboured, and there was a clear mucous discharge from the nostrils. He was destroyed *in extremis*. *Post-mortem*: the only distinctive lesions beyond emaciation were in the liver. This was about normal size; of mottled yellowish-blue colour; lobulation distinctly seen through capsule; the cut surfaces were brick-coloured, and showed distinctly the pale peripheries and distended central veins of the lobules. The gall-bladder was somewhat distended with turbid green bile, and its mucous membrane was injected. Microscopically, practically the whole of the parenchyma was necrosed; only at the peripheries of the lobules could recognisable parenchyma cells be seen.

This beast did not survive long enough after the development of symptoms of acute poisoning to allow me to test its liver function again with phenoltetrachlorphthalein, but to another ox which had been poisoned by five ordinary doses of tartar emetic I gave a test dose of the dye. An hour later its serum was very dark coloured, and alkalinisation showed that more than 10 per cent. of the dye was still in the blood-stream. When destroyed next day its liver was found to be necrosed, though not to the same extent as that of the previous beast.

Since 1925 I have met with this condition of liver necrosis following tartar emetic administration so frequently that I now possess the case-sheets and *post-mortem* reports of twenty animals which have succumbed to it, and I have

seen many other cases of which I do not possess such full particulars.

Of these twenty, sixteen died during their first course of treatment, and four during their second course. The intervals between the injections were from one to seven days, and the time that elapsed between the releasing, or fatal, dose, and death was twelve hours to seven days. The intensity of the necrosis bears no relation to the amount of the drug received nor to the number of days after injection that death occurs. Of course, in the three cases where death occurred within twenty-four hours of the injection it may have been hastened by the drug's toxic action on the respiratory and circulatory systems, but as the livers of two of these showed extreme necrosis there is really no need to postulate this additional factor; what really happens when death follows quickly after a second or subsequent injection is that this merely greatly accelerates the dissolution started by a previous injection.

Symptoms of liver necrosis are fairly characteristic. An affected beast stands by itself in a dejected attitude; with drooping ears, dry muzzle, hollow flanks, harsh coat and staring eyes. Small, hard, mucus-coated balls of dung are passed occasionally and often with straining. Temperature is normal or subnormal. Respiration not appreciably affected. Pulse generally imperceptible, but auscultation may show a rapid, tumultous heart action. Visible mucous membranes are pale, cyanosed, and only rarely icteric. After a time the beast stumbles and falls, and convulsions may occur just prior to death. The patient is not necessarily anæmic. I have several records of animals whose red-cell counts were above normal just before death. The white cell picture varies, as the poisoning is generally of a beast already affected with a blood disease, but if there is a typical picture I should say it is one of leucocytosis with relative and absolute monocytosis and relative and absolute eosinopenia, *e.g.*, white cells, 18,000 per c.m. neutrophiles, 54 per cent.; monocytes, 37 per cent.; lymphocytes, 8 per cent.; eosinophiles, 1 per cent.

If the development of symptoms is not too rapid, and antimony treatment is discontinued, recovery may occur. The first essential of treatment is to overcome the constipation, and for this magnesium sulphate is indicated. Afterwards careful nursing is necessary. If the animal has been cured of his trypanosomiasis he will recover slowly, but if he relapses to trypanosomiasis his case is hopeless, as antimony therapy is now rigidly contra-indicated, and no other drugs can be expected to effect a cure where antimony has failed and when a beast is only slowly recovering adequate liver function.

Post-mortem: the only constant lesions are in the liver. There may be lesions due to trypanosomiasis and intercurrent affections, and the kidneys may show a high degree of fatty degeneration due to the antimony itself, but the lesions which are associated with the direct cause of death and of the chief symptoms just prior to death are confined to the liver.

To the naked eye the liver appears of normal size, or somewhat smaller than normal. The surface colour is always more or less mottled; generally the prevailing colour is a brownish-blue, often with irregular areas of a lighter chocolate tinge mottled with purple. Lobulation is seen distinctly through the capsule. On section a good deal of blood escapes from the cut surfaces. These are of a colour which varies from mottled chocolate to bright ochre. The lobules are distinct owing to their pale peripheries and purple centres around distended central veins. Consistence is firmer than normal. The gall-bladder is generally somewhat distended, but shows no constant pathological changes, nor is the bile usually appreciably altered in appearance.

The microscopical appearance of sections stained with hæmatoxylin and eosin varies, of course, with the degree of degeneration. One can cut pieces of tissue from the same organ which show considerable difference in the degree of degeneration, and even in one section the lobules are not uniformly affected, though generally nearly so. The picture seen is typically that of necrosis starting from the central part of the lobule. In earlier stages only the central parts of some lobules are involved, but in advanced cases the whole of almost all lobules is affected. In the very earliest stage, seen in animals which have died a few hours after a single overdose of antimony, the parenchyma shows only cloudy swelling, the cells being swollen, granular, and lightly stained. In the stage which follows this, one sees a distended central vein, and around this a pink débris of cytoplasm studded with pycnotic nuclei and intersected by apparently intact capillaries, and around this again some peripheral cells which are still intact. Between the intact cells and the necrotic area are some cells which appropriate staining shows to be in a state of fatty degeneration. Although essentially central, yet this necrosis may traverse the periphery of a lobule to connect with the necrotic portion of a neighbouring lobule. (Fig. 1.) In a still more advanced stage the necrosis may involve the whole parenchyma. Sometimes the walls of the central vein break down, and the centre of the lobule becomes flooded with blood. Some sections show a good deal of pigment, but in most it is not conspicuous.

Apparently the necrosis is of the parenchyma alone, and this permits early attempts at repair. In cases where death has taken place more than a day or two after the last injection, one can see that much of the necrotic material has been removed, leaving a framework of capillaries to support the column remnants and any leucocytic infiltration which may be present. It is probably by the rapid mitotic division of these surviving parenchyma cells that the columns are restored in cases of recovery. A conspicuous feature of a picture of early regeneration are the young bile ducts which sprout into the lobules from the periportal tissue. (Fig. 3.)

When sections are stained with osmic acid it can be seen that the cells which lie between the necrosed and intact zones are packed with fat granules. In the necrosed areas the darkly-staining material is in the form of structureless masses and free granules. (Fig. 4.)

There is nothing specific to tartar emetic poisoning in the course which the necrosis follows. For example, it appears to be precisely similar to that which occurs in carbon tetrachloride poisoning, the pathological histology of which has been so ably described by GARDNER and his fellow-workers (1925), and to simulate the acute yellow atrophy of human pathology, as described by MUIR (1926, p. 472.)

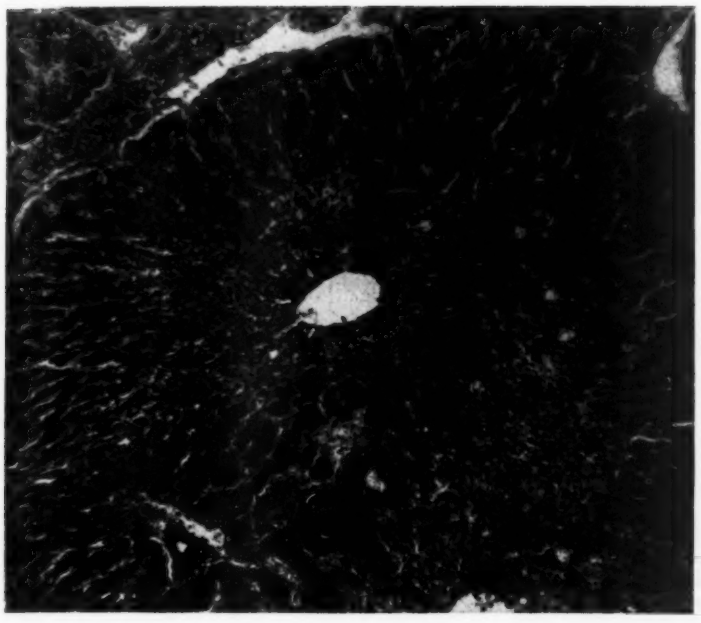

FIG. 1. Central Necrosis, and strand of necrotic tissue connecting two liver lobules. Hæmatox. and Eos. Stain.

FIG. 2. Central Necrosis of lobules—at middle left and top right of figure—and intact portal canal. H. and E. Stain.

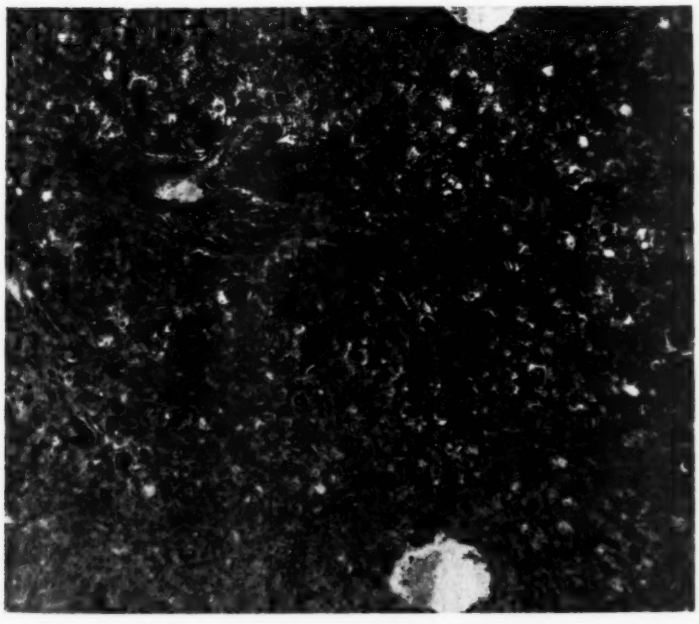

FIG. 3. Nearly complete Necrosis of parenchyma, with some formation of new bile-ducts at peripheries of lobules. H. and E. Stain.

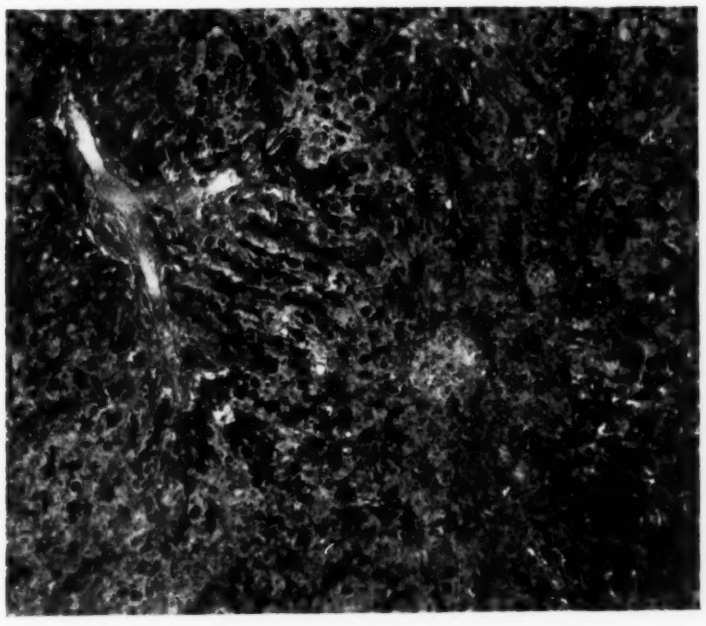

FIG. 4. Nearly complete Necrosis of parenchyma. Osmic Acid Stain.

XUM

It is because the syndrome resembles that of carbon tetrachloride poisoning that I noted with interest the observation made by DAVIS (1924) that a liver well stored with glycogen is more refractory towards large doses of carbon tetrachloride than a liver deficient in glycogen. This is in agreement with the evident fact that a cachectic animal is very much more susceptible to tartar emetic poisoning than is a lusty beast. For example, I picked out two oxen in excellent fit condition, and to each I gave eight doses of tartar emetic, at five and seven-day intervals respectively, without setting up the slightest symptoms or lesions of liver derangement. On the other hand, I have seen extreme necrosis of the liver of an emaciated ox which died the day following a second injection given six days after the first. Nevertheless, I have been unsuccessful in my attempts to fortify a beast's liver by prior injections of glucose.

Recently, I have seen an abstract of an article which appeared in the November (1927) number of the *North American Veterinarian*, in which Dr. M. C. Hall states that prior to treatment with carbon tetrachloride an animal should be put on a diet rich in calcium. As I have not been able to consult the article I am unaware if this is relevant to the subject of antimony poisoning.

It is important that we obtain a better understanding of what constitutes the difference in resistance to the action of dangerous drugs which is shown by the livers of different animals, or by the liver of the same animal under varying conditions of general health. Such knowledge would lead to a method of estimating the adequacy of resistance of any prospective patient, and to the means whereby a low resistance could be raised. Then we should know when therapeutic doses of these dangerous drugs could be safely administered, and when it would be necessary to first treat the liver dysfunction before attempting such therapy.

CONCLUSIONS.

The intravenous administration of even therapeutic doses of tartar emetic to cattle is by no means without grave danger. The most serious toxic action of the drug is on the liver, where it is liable to provoke central necrosis closely resembling acute yellow atrophy of human pathology.

Great differences exist between individual animals concerning their susceptibility to this form of poisoning; cachectic animals being very much more susceptible than lusty ones.

Research is needed, first to reveal on what this difference depends, and then to show how animals with livers of low resistance can be treated so that this resistance may be raised sufficiently to permit efficient antimony therapy.

I wish to express my gratitude to E. B. Verney, Esq., M.A., M.B., Professor of Pharmacology, University College, London, for advice and other assistance in the preparation of this article.

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ARMY VETERINARY SERVICE.

LONDON GAZETTE—WAR OFFICE—REGULAR FORCES.

Jan. 6th.—Lt. A. G. Heveningham is seed. for duty with the Sudan Def. Force (Dec. 2, 1927).

TERRITORIAL ARMY.

Lt. C. P. Johnson resigns his commn. on account of ill-health (Jan. 7).

REGULAR FORCES.

Jan. 10.—Maj. (Bt. Lt.-Col.) J. J. B. Tapley, D.S.O., to be Lt.-Col. (Jan. 9).

MEMORANDA.

Col. A. G. Todd, C.B.E., D.S.O., late R.A.V.C., having attained the age fixed for compulsory retirement, is placed on ret. pay (Jan. 9).

The follg. Lt.-Col. to be Col.: A. Olver, C.B., C.M.G., F.R.C.V.S., R.A.V.C. (Jan. 9).

R.S.P.C.A. *versus* Ministry of Agriculture.

ALLEGED CRUELTY TO ANIMALS AT SWINDON.

The following report is reproduced from the *Swindon Evening Advertiser and North Wilts. Herald* of Tuesday, January 3rd :—

Two inspectors of the Ministry of Agriculture, Arthur Holman Berry and Albert Victor Franklin, were prosecuted by the R.S.P.C.A. at the Swindon County Police Court to-day for causing unnecessary suffering to a number of cows on 6th November. The case, which aroused considerable public interest, arose out of the slaughter of cattle at Westlecott Farm,Swindon,onthe occasion of the recent outbreak of foot-and-mouth disease.

The magistrates were General T. C. P. Calley, C.B., M.V.O. (chairman), Major Cecil Wilson, Messrs. F. A. Blake, W. Dale and A. W. Deacon.

Mr. R. Mitchell Banks, K.C., M.P. for Swindon, with whom was Mr. F. Wishart, appeared for the prosecution, and the defendants were represented by the Hon. Jeffery Laurence, K.C., with whom was Mr. H. Maddocks.

Opening the case for the prosecution, Mr. Mitchell Banks said the prosecution was brought by Mr. W. T. Stevens, the local inspector of the Royal Society for the Prevention of Cruelty to Animals, against the two defendants, who were veterinary officials employed by the Ministry of Agriculture and Fisheries. If the bench decided to convict, they would be liable to a fine not exceeding £25, or imprisonment not exceeding three months.

The facts were that in November there was an outbreak of foot-and-mouth disease on the farm of Mr. Arthur Balch, at Westlecott. The farm was taken over by the officials of the Ministry of Agriculture on 5th November, and the slaughter of the animals was arranged for. Trenches were dug at about 200 yards distance from the cow sheds, and the slaughter took place on 6th November. This was seen by Mr. T. A. Sutton, who was in his grounds about 400 yards away, and used a pair of field glasses. He called Mr. Stevens and the police.

Mr. Banks said that the cows were taken from the sheds to the trenches, where they were slaughtered. At first two or three animals went quietly, but later, as Mr. Sutton would say, the others became terrified. There was no question about the method of killing, as the humane killer was used, but what they objected to was the dragging of the animals behind carts, terrified, and resisting.

In some cases the animals were being struck, continued Mr. Banks, and were physically suffering, while others fell down as they were being pulled along by the cart, sixty-three cows were treated like this in the morning, fifteen heifers and two bulls were killed outside the shed in the afternoon, in a proper manner. When interviewed about the alleged cruelty in the morning, Mr. Berry said the method was the one generally adopted in Great Britain, Scotland, and Ireland. The men who did the slaughtering were experts, and he and Mr. Franklin had given orders that there must be no cruelty. The evidence would show that the animals suffered both physically and mentally. The method adopted was the most dangerous one, so far as spreading the disease was concerned, for when the animals were in this terrified condition, more saliva came from their mouths than usual. If the animals that were killed in the morning had been killed in the shed in the same manner that others were killed in the afternoon it would have been far better. He (Mr. Banks) would suggest that the methods adopted were far from being wise and prudent in the prevention of infection. In the afternoon the animals were slaughtered in the sheds. There was no complaint about the actual killing of the beasts, which was done by the use of a humane killer. There was no allegation of fiendish and deliberate cruelty, but the prosecution suggested that unnecessary cruelty was caused to the cattle by the manner in which they were killed on the morning of Sunday, 6th November.

AN EYE WITNESS'S STORY.

Thomas Sutton, agent for Mrs. Goddard, of the Lawn, Swindon, was the first witness. He said that on the morning of 6th November, he was in his garden at his residence at Westlecott. He saw cows being dragged by means of a cart, to which they were attached by ropes. They were taken by a circular route and not direct from the cowsheds to the trenches—a distance of 250 yards or more. He saw between twenty and thirty cows dealt with.

Mr. Wishart : Were the cows going willingly ?

Witness : No, they were not. I saw one fall down, and it was hit with a stick, and dragged along. There was shouting and hooliganism going on. He had had many years' experience as a farmer, and in his opinion the cows were suffering greatly from the method adopted. Witness reported the matter to Mr. W. T. Stevens, the local Inspector of the R.S.P.C.A., and to the police, and Mr. Stevens arrived, and also the Deputy Chief Constable (Supt. Brooks) and Inspector Mitchell.

Cross-examined by the Hon. Mr. Laurence, Mr. Sutton said he was 300 or 350 yards from the spot. There was not a belt of trees immediately between his house and the place where the cattle were taken to be slaughtered to obscure his vision.

Mr. Laurence : Is it true the cows were led windward so as to avoid the smell of blood ?

Witness : They were led in an up-wind direction. The wind was fairly still, and my vision was perfect. The cows were led along in a manner like we would park motor cars.

Replying to further questions put to him by Mr. Laurence, witness said he had not heard why the cows were tied behind a cart when they were led to slaughter. He did not agree that it was done in order to prevent each cow seeing the actual killing of another. He did not think that was the reason why they were tied and dragged behind a cart.

Mr. Laurence : Did you say that cattle are not frightened by the view of the slaughtering of other animals ?

Witness : No, in my opinion the sense of smell is the chief thing with animals—and noise. Witness added that he considered that a temporary slaughterhouse should have been erected, and the animals killed there one at a time. That could be done where the spending of money was no object.

Mr. Laurence : Do you mean to say you come to this court and tell us that money is no object, when the cost which is entailed in these outbreaks falls on the taxpayers and ratepayers ?

Witness : Well, it seems so.

Would you suggest that a temporary slaughterhouse should be erected in every case where there is an outbreak of foot-and-mouth disease ? Yes, I do.

Answering further questions, Mr. Sutton said three horses and three carts were being used in the dragging of the cows, which were each attached in turn to a cart by means of ropes. When Inspector Mitchell arrived, witness spoke to him, but the Inspector did not say he had seen any cruelty. The Deputy Chief Constable (Supt. Brooks) did say he had seen no cruelty.

Inspector Stevens, the local inspector for the R.S.P.C.A., said that at 10 a.m. on Sunday, 6th November, he went to Mr. Sutton's house in response to a call. He saw twenty men on Westlecott Farm, with three horses and three carts. Each horse and cart in turn was backed to a cow-shed door, and an animal tied at the back. They went round the cow-shed into a grass field adjoining the yard. He noticed several cows showing signs of great distress. He saw one fall, and a tall man hit her when down, either with a stick or strap. As each cow got to the trench they were killed remarkably well by a humane killer. The wind was blowing from the carcases towards the cow-sheds, and would meet the cows as they were coming out behind the carts. The men were shouting, and one said : " Oh, to be a farmer's boy," and appeared to be making fun of it.

Inspector Mitchell arrived later, and Supt. Brooks went to the farm. When they arrived, the shouting ceased, and more order was restored. He interviewed Mr. Berry on 7th November, and he said that it was the general method adopted throughout the country. They did not want any animal to break loose and spread the disease.

On 17th November, he again saw Mr. Berry and informed him that he would report the case.

Cross-examined, witness said one man struck a cow with a stick, and he believed that this was cruelty. Witness saw Mr. Berry twice on 7th November, and twice afterwards, not nine times, as was contended. He asked Mr. Berry if the cart was put there to shield the cows from seeing dead animals. If that was the reason, he said it was a very good point on their behalf, but he got no answer. Mr. Berry asked him if there was more cruelty at the farm than in slaughterhouses, and witness suggested that there was.

Oliver David Stiles, farm labourer, of 1 The Quarries, Swindon, said he was employed by Mr. A. Balch, farmer, of Westlecott. On the morning of 6th November he dug some trenches on the farm. He was in charge of the grey horse and cart, and took the first cow to be slaughtered. Two cows fell down behind the cart. He took them as near to the trench as possible, so that they would fall into it when slaughtered. Sixty-three cows were taken in this way. Sixteen animals were killed outside the shed in the afternoon, because they were young animals. As he was driving he could not see the cows hanging back, but could see this behind other carts.

Cross-examined, witness said if a cow fell down, the horse would ease up because of the weight. The cows were not tied up too tight by the chains, and the rope on their horns was a safety line. There was nothing cruel or brutal in what he saw done that morning.

Replying to Mr. Banks, he said the cows taken out of the shed in the afternoon suffered less, because they did not have far to go.

Sidney John Lambert Ferris, farm labourer, of 5 Okus Cottages, said that on 6th November he was on the farm when the animals were slaughtered. He had the brown horse and cart and took the cows to the trenches very well. The rope over the cow's horn was for safety purposes, and was held by three men.

Mr. Banks: Surely the chain wouldn't break to need a rope?

Witness: No, but safety's best. (Laughter.)

A cow fell down behind his cart on the journey, added witness. Cross-examined, witness said he had been on the farm nine months. He did not think the cows were treated cruelly. He was one of the men instructed on the Saturday by Mr. Berry, who said they must carry out the rules. He did not say the cattle were to be dragged behind carts.

William Dowling, market gardener, of 17 Quarry Road, Swindon, said at 9-20 a.m. on 6th November he was on the allotments near the farm. He saw the cows being pulled out from the shed, and they were not going willingly. Later he was under the bridge, and saw the cows being dragged along unwillingly.

Cross-examined, witness said he did not see any cows on the ground. Animals did not as a rule hang back like these did. The animals went between 200 and 300 yards detour from the sheds to the trench.

Thomas Havelock Higgs, farmer, of West Leaze, Swindon, said that at 9 a.m. on 6th November he was in his garden with his wife. Looking across to the farm he saw cattle being dragged behind a cart. The cows were not going at all willingly.

Cross-examined, witness said he farmed 50 acres of land, and his farm skirted that of Mr. Balch. He was 600 to 700 yards from the farm buildings. He saw several cows hang back, but none on the ground. He approved of the method of slaughtering for foot-and-mouth disease.

Alexander Piesse, M.R.C.V.S., of 277 Brixton Road, London, said he had been in practice for forty years. He thought the way the animals were slaughtered in this case would lead to increasing the infection. Salivation would be increased, and a wind would broadcast the saliva. He thought the animals should be killed near the shed, and taken on a trolley to the trenches.

Cross-examined, witness said he was the official veterinary surgeon to the R.S.P.C.A. He had not had much experience of foot-and-mouth disease in the last twenty years, but at his College had studied it. He believed that time was of great importance in dealing with such diseases, and in this case he thought more horses should be brought in from outside. They could be properly disinfected. He agreed that there was a fear among cattle when brought to the slaughterhouse, but anything of this should be avoided when possible.

This closed the case for the prosecution.

Case for the Defence.

In opening the case for the defence, the Hon. Jeffery Laurence said the two defendants, who were veterinary surgeons, were in the employ of the Ministry of Agriculture, and were permanent officers of the Ministry. If it were not for the fact that this case was one of great national importance, he might not have thought seriously of submitting that this case was one where there had been no cruelty. He was proposing to call a number of witnesses, including representatives of a body of public opinion unequalled in England on the subject. In addition he had as witnesses nearly all the men who were present on the farm on the day of the alleged offence. The two men working on the farm who had been called as witnesses for the prosecution had both said they saw no sign of cruelty. He was not going to suggest that Mr. Tom Sutton, in instituting this prosecution, was not actuated by humanitarian motives. But in matters of this sort, the magistrates, with their great experience, knew that there were different people taking different views. Some people were hypersensitive.

In the present case the bench had before them a veterinary problem of great importance. It was a case of great importance to the people of England as a whole, and to the farming industry in particular, that this disease should, if possible, be stamped out. What the magistrates had to consider was not whether pain was inflicted, but whether pain was unnecessarily inflicted.

Referring to the position of Mr. Berry, counsel said it was a serious matter for a man with Mr. Berry's long career and experience that he should be brought to the court and placed in the dock, so to speak, and charged with this serious offence. Mr. Berry was an official of the Ministry of Agriculture, known throughout the profession for his humane methods. The charge which he had to answer was that of a criminal offence, and he had carried out methods recognised by the Ministry, and evolved after thirty or forty years' experience, in combating this disease.

The suggestion which had been made that a temporary slaughterhouse should be used was a ridiculous one, and the suggestion that the cattle should be carted from the place of slaughter to the place of burial was almost equally ridiculous, because it could not be done. It would be ridiculous to argue before a bench of county magistrates that the evidence which had been given showed that there had been cruelty proved in this case. All cattle taken to a slaughterhouse were led to a place where they could smell blood. Some pedigree cattle were slaughtered on farms, and not taken to a recognised slaughterhouse. It could be done, but it entailed extra expense.

Counsel's Submission.

After further comment upon the evidence which had been given, Mr. Laurence said he must submit strongly that this painful business of killing a large number of cattle for the purpose of endeavouring to stamp out the disease was carried out in the most expeditious way, and in a manner that did not cause unnecessary suffering to the animals.

Col. Young, M.R.C.V.S., Veterinary Officer for the Corporation of the City of London, said he had over thirty years' experience in the biggest markets in England. He had had experience of foot-and-mouth disease, both at home and abroad. In his opinion the essential thing in the prevention of the spread of foot-and-mouth disease was that the animal should be killed as quickly as possible. The method used here was one of the quickest, and it was also adopted in America. Expedition was the great thing.

He thought that if the slaughtering had been done at the yard, and the carcases removed to the place of burial, it would have taken two days. In the incubative stage the blood was the chief source of disease, and so it must be prevented from spilling. To bring horses on to an infected farm was very bad management, and he could not imagine that a farmer would lend his horses for the purpose. It was practically impossible to disinfect a horse because of the hair. On this farm only one cow had the disease, and the others were in contact. Cross-examined, witness said that the distance from the shed to the trench was 120 yards by the circular route. Newly-shed blood did not have a smell at all, said Colonel Young. He had seen animals slain in France with others standing round them, eating, and not paying any attention at all. Knowledge of the prevention of the disease had progressed a lot in the past twenty years, but not as much as it should do. He said that the saliva would not travel very far. Whether it was saliva or blood it would be on the track. They adopted a circuitous route so that the animals could not see the others. In his opinion if they did that to avoid the smell of blood, they were wasting unnecessary time. With reference to the burning of the cattle, Colonel Young said he thought all the fuel required should be on the ground before the infected animals were brought out. Replying to further questions, Colonel Young said the heifers were not taken to the field because they were untrained, and might break their necks.

Professor Hobday, Principal of the Royal Veterinary College, London, said that in this case he thought the method was the most humane one that could be devised. The main objects in the prevention of the disease were to get the slaughtering done as quickly as possible, and to stop as much contagion as possible. He thought it would have been a great risk to bring horses on to the farm, and only a foolish farmer would have loaned them. He knew Mr. Berry and thought he would be the last man in the world to do anything cruel.

Cross-examined, witness said he took the opinion of experts that the microbe in the incubation stage was in the blood and not the saliva. He agreed that the circuitous journey caused extra infection. The roundabout journey was taken to prevent the animals seeing the others killed.

With the use of the humane killer, very little blood would be spilt.

Asked about his experience of foot-and-mouth, witness said he saw the largest outbreak the world has ever known in Italy. He had dealt with the disease in England, and all the countries of Europe. Witness went on to say that it was of the utmost importance to get the animals burnt as quickly as possible.

Arthur Holman Berry, F.R.C.V.S., said he had been a superintending inspector for the Ministry of Agriculture since 1910. He had been an inspector for thirty years, and the method used had been adopted after long experience. It was the best method for dealing with cows, but heifers could not be treated in the same way. He came to Swindon on 31st October, and went to Westlecott Farm on 5th November. He found only one animal affected. He gave instructions to Mr. Franklin, the assistant inspector, to carry out the slaughter. No details of how the slaughter was to be done were given, as the inspectors were fully qualified men. The slaughter took place on 6th November. An anonymous message was received informing him that "dirty work" was going on behind his back, and he later saw Supt. Brooks.

On 7th November, Inspector Stevens visited him twice, and said he was going to take steps concerning the alleged cruelty. Inspector Stevens did not seem to know much about it, and made frequent calls afterwards. Mr. Sutton later called on him, and said he did not approve of the method.

Cross-examined, witness said that the method was an improved one. He liked it because it was quiet and orderly. Other methods were adopted on different occasions.

Replying to the chairman, witness said that this was the fourth outbreak in the district, and he was present. The orders for the construction of the pit were carried out by Mr. Franklin, on his instructions. He did not visit the place on 6th November, but he went there on the day of the outbreak. The orders given to Mr. Franklin were sent to witness by the Ministry of Agriculture.

Replying to Mr. Banks, witness said the Ministry of Agriculture knew what method was adopted.

Albert Victor Franklin, M.R.C.V.S., an inspector employed by the Ministry of Agriculture, said he went to the farm on 6th November. He was responsible for the carrying out of the slaughter, as he made the arrangements. Horses, etc., had to be taken into consideration for the preparation of the slaughter. In view of the facilities available he adopted the method concerned. The cows were quiet, but the heifers were lively. It would have taken two or three days to kill the cows, and take the corpses to the trenches. He commenced the slaughter himself because the slaughterer was not there at the fixed time. He gave instructions to the men that they were to be quiet before the work commenced.

The slaughter commenced at 8 a.m., and finished at noon. By this method the corpses were disposed of in one day. Had the method suggested by Mr. Banks been adopted, the work would have taken more than one day. There was no noise or hooliganism on the farm. With reference to the disposing of the bodies, witness said they were burnt out in from twenty-four to forty-eight hours. The fuel was brought on the farm during the time of slaughter and afterwards. None of the cows resisted going to the trench. He had never seen cows walk so well before. They were put behind the cart for the purpose of control. The circuitous track was for the sake of getting the animals in position, and not to prevent the smell of blood.

Replying to the chairman, witness said he was in charge of the operations. All the instructions he had got were to get on with the job and slaughter the cattle. Everything that was done was by his direct orders. No orders were issued by the Ministry of Agriculture.

The Deputy Chief Constable (Supt. Brooks) said he went to the farm at 10-30 a.m. on 6th November, whilst he sent Inspector Mitchell to the residence of Mr. Sutton. He saw the remainder of the cows slaughtered, and thought that the work was carried out in the most humane manner. Not more than two minutes elapsed between the time the cows were brought out of the shed till they were slaughtered. He saw no cruelty whatever. There was plenty of room to slaughter the cows in the yard, but great difficulty would have been encountered in getting the dead bodies to the field.

Cross-examined, witness said that Inspector Stevens saw him some days afterwards, and discussed the matter with him. Inspector Stevens and Mr. Sutton were inaccurate in saying that there was resistance, as there was none when he was there.

Inspector Mitchell said that on 6th November he went to Mr. Sutton's house, and he and Inspector Stevens looked on the proceedings for an hour and a half. He saw no sign of cruelty during that time. When commenting upon this, Mr. Sutton said "What would you expect now that the Deputy Chief Constable is down there?"

P.C. Clarke said he was on duty at Westlecott farm early on Sunday morning, 6th November. He saw some thirty head of cattle taken out of the sheds and led to slaughter. He saw no animal resist or fall down. He saw no form of cruelty and heard no shouting. He thought those witnesses who had said there was shouting and cruelty must have been mistaken.

Capt. G. Atkinson, Inspector of the Ministry of Agriculture, said he went to Westlecott farm at 10-30 a.m. on 6th November, on receiving orders from Mr. Berry. He saw no cruelty or beating, nor any cows fall down. Twenty were lead out while he was there. The method employed was commonly used, but it depended upon the size of the buildings and the number and size of the cattle.

Cross-examined, he said he did not see the animals killed in the afternoon. The bulls killed in the shed were heavier than cows, but he did not know how long it took to get them to the trenches.

Ernest William Lanfear, foreman at the farm, said two horses were used to pull the bulls, and it took then twenty minutes each to get to the trench.

Cross-examined, witness said it would not have been any quicker to have dragged the bulls on a skip.

Mr. Laurence indicated that he had all the men who were engaged on the farm at the time in Court to give evidence if the Bench required to hear them.

The Chairman (General Calley) intimated that they did not require more evidence.

The Bench then retired for consultation, and were only absent five minutes. On their return the Chairman said: "We have given careful consideration to this case. We have heard a number of witnesses, and we are of the opinion that the evidence does not bear out the allegation that there was any unnecessary cruelty used. We therefore dismiss the case. We are of the opinion that there were sufficient grounds for bringing the action, and consequently we make no order as to costs. Each side must pay its own."

The Third World's Poultry Congress.

"The idea of holding an International Poultry Congress originated with Dr. Edward Brown some time before the year 1914, but, before plans could be completed, the Great War temporarily arrested all development in this direction," says the *Journal of the Ministry of Agriculture*. "On the conclusion of hostilities, the project again came up for consideration, and it was agreed that, in view of existing conditions, the inaugural congress should be held in one of the non-belligerent countries. Accordingly, on the invitation of Her Majesty the Queen of Holland, the first Congress was held at The Hague during the summer of 1921, and three years later the second Congress took place at Barcelona, under the patronage of the King of Spain and the Prince of the Asturias, both of whom gave it their active support.

"The third World's Poultry Congress, held at Ottawa in the Dominion of Canada from July 27th to August 3rd last year, was attended by some 3,000 delegates representing 43 countries, and must be regarded as one of the outstanding events in the history of the industry. The Congress was organised in five sections, and considered 150 papers submitted by specialists in the several branches of research, instruction, and marketing. The Exhibition in connexion with the Congress proved an extremley popular feature, being visited by at least 140,000 persons. Apart from an extremely fine display of live birds from many countries, of which 350 were sent from this country, the Exhibition was arranged in national sections, and consisted mainly of educational and scientific exhibits. The British section comprised items illustrating diseases, breeding, and nutrition, and pictures and models relating to various phases of the industry. Some valuable pigeons and poultry belonging to His Majesty the King and H.R.H. the Prince of Wales were also included. The exhibits displayed by the Canadian Departments of Agriculture, both Dominion and Provincial, were naturally prominent, but it is satisfactory to note that the British section also attracted much attention, and was favourably commented on by many of the delegates.

"The Right Hon. Stanley Baldwin attended a special meeting of the International Association of Poultry Instructors and Investigators for the purpose of conveying the invitation of the Imperial Government to hold the next World's Poultry Congress in Great Britain. The Congress, he said, could be assured of the support of the British Government and the Ministry of Agriculture, and a cordial welcome from the numerous poultry organisations in this country. As this invitation was unanimously accepted, the next Congress will be held in Great Britain in 1930."

"Grass Disease" in Horses.

Discussion on Papers by Professors Pool and Greig at N.V.M.A. Northern Branch Meeting.

Professor W. A. Pool: Before the discussion commences, I would like to say a few words. When I was asked to write a paper for discussion at this meeting, I thought that "grass disease" would be a good subject to choose because it is of such great importance in Scotland, and would ensure an interesting debate. In addition, it is expedient at this stage in the investigation to place on record a review of the literature dealing with diseases which may or may not be allied to "grass disease." An attempt has therefore been made to summarise the important publications bearing on this question, and then to discuss the relationship of "grass disease" to these conditions.

At present the cause of "grass disease" must be considered to be an open question. There is no justification on the evidence available to form any one theory and support it strongly.

The urgent need now is for team work in the investigation, enlisting the help of specialised scientists whose work may have a bearing on the problems involved. We are fortunate in that these scientists are interested in the subject, and willing to give all the help they can. We have here to-day Professor Clarke, Dr. Wilson, and Dr. Dryerre, of Edinburgh University, and will be interested to hear their views.

The work at present in progress in the laboratory of the Animal Diseases Research Association is not discussed because it is still incomplete.

No mention is made of the morbid histology of the tissues because it has not yet been adequately studied. Material has been collected and the results of its examination will form the subject of a subsequent publication.

A great deal of work has been carried out on the bacteriology of "grass disease." A great deal of it has yielded negative results. The existence of certain cocci in the central nervous system described by other investigators has been confirmed, but they appear to be only of secondary importance.

Professor Russell Greig: I do not think that I can profitably contribute to the discussion at this stage.

As a result of a study of the clinical symptoms of acute grass disease I had formed certain opinions as to how these symptoms might be properly interpreted, but when I was invited to express my views at this meeting, I accepted the invitation with a certain reluctance, as my conclusions have been reached by a process of reasoning, and as yet are almost wholly unsupported by experimental evidence.

However, I submit the paper to you in the hope that, if it has no other value, it may not be without interest to the practitioner, since the study of the correlation of clinical symptoms with inbalance of the involuntary nervous system is new to veterinary medicine, and I believe that a clearer conception of such correlation may shed light on the nature of several conditions, other than grass disease.

Mr. W. Nairn (Blairgowrie): I feel sure that many, when they received their copy of Professor Pool's paper, would open it with considerable interest to learn what striking discovery had been made in this disastrous equine disease. All, I am certain, will wish Professor Pool an early and successful termination to his labours in this disease, and give him every assistance in their power to attain his purpose.

To a practitioner it would seem that the usual and recognised methods of research have so far yielded no result, and that possibly some modification of method may be required. Professor Pool, I think, has acted wisely in bringing the results of the investigation up to date, and in giving us a resumé of world literature dealing with diseases which some have held to be similar to "grass disease." This study is interesting both from the point of view of research and from the clinical aspect.

As you have seen, he discusses epizootic cerebro-spinal meningitis, forage poisoning, Borna disease, and botulism. A survey of American literature suggests that epizootic cerebro-spinal meningitis and forage poisoning are the same disease, while Hutyra and Marek regard Borna disease and epizootic cerebro-spinal meningitis as similar. We may therefore conclude that these diseases are all related, and that the nomenclature depends on the location. Graham and Himmelberger arrived at the conclusion that forage poisoning was botulism, so we add another to the list of synonyms. These workers, when studying forage poisoning, were able repeatedly to produce the disease by feeding, and this brings it within the increasing list of ailments produced by the ingestion of toxins in the food.

I find it rather difficult to form a very clear picture of these diseases from modern textbooks, but it would appear that the account of any one would fit any of the others mentioned. The symptoms given by Graham for botulism and forage poisoning, and by Hutyra and Marek for Borna disease and cerebro-spinal meningitis might all be summarised in the old-fashioned but descriptive title of "staggers." They all show little disturbance of temperature but varying disturbance of pulse. There is an inclination to press forwards: soon a lack of muscular control with early collapse on the floor. But for occasional spasms of excitement when disturbed, which recall the old "mad staggers," the usual drowsy dulness gradually increases until the animal is comatose.

While including Borna disease, I feel that Hutyra and Marek's picture of a case is not very clear: they mention muscular spasms, excitement, and early paralysis, and say that while in some cases cerebral symptoms are the most outstanding, in others bulbar symptoms predominate, and a few show only spinal manifestations. I have never seen an acute case of "grass disease" showing cerebral symptoms, but perhaps it would be better to define what I regard as cerebral symptoms in the horse. They are dulness, drowsiness, with fits of excitement when disturbed: a desire to press forward, and usually some disturbance of co-ordination. As symptoms of bulbar paralysis authors mention difficulty in swallowing in every case: the animal chews but cannot swallow: secretion of saliva is increased: later, the animal cannot drink, the muscles of the face become relaxed, the tongue is paralysed, and at length the muscles of mastication lose their function.

At the beginning of their description of symptoms they give an account of features which, if the picture ended there, might suggest a relationship with "grass disease," *viz.*, loss of appetitie, jaundice, colic, catarrh of the pharynx, with increasing depression and dulness which continues to the end, but they speak of attacks of vertigo, excitement and mania, and these I have never seen in "grass disease."

This description of botulism by Graham is somewhat different from the picture of botulism in horses given by Theiler and Robinson in Africa. They classified the types of cases as peracute, acute, subacute and chronic. In the peracute the animal was usually dead or dying: when found alive it was unable to rise, and often comatose: the recumbent animal, before the comatose stage develops, frequently struggles to rise but merely gets partially up and falls back again. In this form there is no suggestion of any bulbar disturbance. An acute case may continue for two days and during that time the animal shows increasing weakness of muscles, eventually falling down and, like the previous type, indulging in fruitless struggles. At the beginning of the case bulbar symptoms may be seen, but latterly the locomotor paralysis is the outstanding feature of the case. In the subacute form the symptoms are more prolonged, and the final stage may not be reached for six days.

The chronic form may only show a stumbling, unsteady gait, which may in some cases reduce the victim to a recumbent position, when it may have to be destroyed, but many recover after weeks or even months.

Drinking of water and swallowing of food are both difficult in the acute and subacute forms, and often the first striking symptom is the paralysis of the tongue, which hangs from the mouth. In the chronic form the animals do not lose their appetite and the activity of the bowels is not disturbed in the most of the cases. An odd case shows a degree of constipation.

While we are considering botulism in the horse it might be interesting to consider briefly botulism in other animals, and in all the forms recorded the outstanding feature is the great muscular weakness. In lamziekté, as described by Theiler, the outstanding symptoms are the great weakness, with more or less rapid collapse, and the paralysis of the tongue. In limberneck in fowls we see a similar muscular weakness, so much so, that the muscles of the neck cannot support the head: the weakness increases until the animal is unable to control any of its voluntary muscles. In parabotulism of cattle, as recorded by Seddon in Australia, the same muscular weakness holds, with a similar paralysis of the tongue.

In their studies of forage poisoning Graham and Himmelberger found in their experimental animals a similar train of symptoms, exhibited both by horses and cattle, but they found that cattle had more resistance than horses: whereas Theiler, when experimenting with his lamziekté toxin, found horses more resistant than cattle, and fifty times more so. Both species would seem to be susceptible to botulism, but their resistance varies. In the "grass sickness" area we have no record of cattle dying from any obscure disease at the same season as we find the heavy mortality in horses.

In making a comparative study of the symptoms of the diseases already mentioned, we note that the marked difference between "grass sickness" and the other group of diseases already mentioned, *viz.*, Borna disease, cerebro-spinal meningitis and botulism is that "grass sickness" is the only one in which the locomotor centres are not affected. This is a clearly-marked distinction which is not clouded by any intermediate type of case. In "grass sickness" and in the others we see features which may be ascribed to bulbar disturbance, but we never see in "grass sickness" any marked loss of intelligence, any marked drowsiness passing into coma, nor any paralysis of the tongue.

All authors describe in Borna disease and forage poisoning the pressing forward symptoms, blindness, stupor and drowsiness, which give the syndrome of "staggers," at times "mad staggers." Constipation or intestinal stasis, which is such a marked feature of "grass sickness," is usually noted in all these diseases, but it does not receive the emphasis which we are accustomed to give to it in "grass sickness."

I need not here recapitulate the outstanding symptoms of "grass sickness," as Professor Pool has given us a summary which is almost as illuminating as a paragraph from Hutyra and Marek on the symptoms of Borna.

I should like for a minute to draw your attention to the various hypothetical discussions on the etiology of "grass sickness." Professor Pool gives us no account of the histo-pathology of "grass sickness," which seems to me to be a serious omission; perhaps he will tell us whether or not he found any evidence of encephalitis. This point has never been satisfactorily cleared up, and it might help to show whether there was an actual infection of the brain cells. He is very guarded in the expression of his views on the etiology, and while submitting the various hypotheses which have been advanced, he does not indicate the one he favours most. A practitioner need not be so reticent in the expression of his views on this subject, and I propose to discuss from the clinical point of view the various hypotheses, beginning with the poisonous plant theory, as it is one of the oldest.

When the mortality appeared only while the animal was at grass it was but natural to suspect some plant as the source of the poison, but the various surveys of pastures in the area have failed to incriminate any plant.

The high incidence for a few weeks in the year is not in favour of the view, nor is the appearance of the disease on a farm one year and its absence for several years afterwards, although the pasture is unchanged. We

find it on hill grazings and again on rich arable fields with no relationship in the vegetation grown. Botanical surveys have so far accounted for the safety of all the common plants found on arable fields where the disease was prevalent, but we have no knowledge of the microscopic vegetation which may exist on plants with deleterious actions. There is no strong evidence that cases arise from feeding hay from a field on which grass disease was prevalent the following year.

At different times various organisms have been charged with the incidence, and we have had streptococci, bacilli, and filter-passing organisms all in turn held responsible. It would be rather unusual if pathogenic organisms entered in the blood stream or the nerve centres and produced no increase of temperature in the early stages of infection.

Many may not agree that there is no increase of temperature in the early stages, but I have records of cases which showed on a first visit no increase of temperature or pulse, only some abdominal discomfort. In a few hours the pulse was much accelerated and the temperature raised with a fatal termination in twenty-four hours. I am inclined to think that the temperature and reaction in "grass disease" are due to the secondary metabolic disturbance and not to any initial systemic invasion.

We have been told that these various organisms produce meningitis and encephalitis, but if the disturbance of nervous function is due to the invasion of the nerve centres, why does not the invasion of these organs advance to a stage when it would be evident that clinically we were dealing with a disease of the central nervous system? We might expect such symptoms as spasms, paralysis of groups of muscles, a tendency to press forwards, excitement, alternating with drowsiness and, later—coma. Borna disease seems to be a disease of this type, and in it we see signs of a progressive pathological process.

Professor Pool states that for the filter-passer a reservoir of infection is required, but he does not make it clear whether he would expect that there were carriers among the horses, or a large number of infected flies in the neighbourhood. If any equine carrier was responsible we would expect cases among horses in the stable rather than at grass. If spread by biting insects, we must account for cases which occur at seasons when flies are absent, as in December.

Horses working in a fly area would be as likely to get infection as animals at grass, since they do not escape the attentions of the various biting flies.

In the late spring, examination of the insides of ears and the thin parts of the skin on the abdomen will show evidence of insect bites and that, on farms where several months later cases of "grass disease" are seen, but there can hardly be any connection between these early insect attacks and the outbreak of disease.

Various cocci have been put forward as the causal agents, but generally the proof has not been completed and various workers to-day hold that their part is at most secondary.

The other hypothesis mentioned by Professor Pool is our old friend botulism. As shown earlier, the whole of the world literature dealing with botulism describes symptoms we never see in "grass sickness," *viz.*, marked muscular weakness with loss of co-ordination, and usually associated with paralysis of the tongue and lower jaw.

Forage poisoning, lamziekté, parabotulism of Seddon, botulism of horses described by Theiler and Robinson, and limberneck in fowls all provide these features to a well-marked extent.

Professor Pool appears interested in Theiler and Robinson's theory that the bodies of dead animals, such as rats, can contaminate the food. I hardly think there is much risk of this on arable grass, but we would expect to find more cases in horses when fed on hay and oats as it is no uncommon experience about a farm to find these rodents dead among hay during the winter season.

The horse is not an eater of garbage, and if dead animals played any part we would be more likely to find that cattle were the victims.

There is one constant feature in an outbreak of "grass disease," as suggested by the name—that the animal must be fed on grass. It does not matter whether it is outside or inside the stable, but the outside would seem to be the more risky.

The animals show no disturbance of pulse and temperature if seen early, but these disturbances are soon apparent as the case progresses.

There is no evidence of any extension of the destruction of the nervous function. The amount of disturbance seems to be determined at the commencement and probably depends on the amount of toxin ingested; any aggravation of symptoms depends on the general interference with metabolism and the amount of interference is seemingly determined by the quantity of toxin ingested.

What, then, is the source of the toxin? We were told at one time it was due to the *Bacillus botulinus B.* but, as pointed out earlier, other workers in various parts of the world who have recorded their findings give a picture of botulism which is very different from "grass sickness." One notes that some authors speak of a toxico-genic saprophyte causing some of the diseases we have discussed, but I always understood a saprophyte as living on decaying matter. If "grass disease" depended on decaying vegetable matter we would hear less of it. Then, can there be any other type of vegetative organism which can be toxic when ingested? I am inclined to think there is an organismal growth on grasses which takes place in parts favourable to its existence, and that weather conditions and the stage of ripeness of grass modify its toxicity.

Discussing the interpretation of the symptoms of "grass disease," Professor Greig, in a very worthy effort to throw light on the physio-pathological changes in the disease and which changes are reflected in the clinical features we are familiar with, has suggested that the symptoms arise from stimulation of the sympathetic centres. If that is correct, would it not require a constant supply of toxin being manufactured to keep up this reaction? Otherwise, a cure would soon follow the exhaustion of supply. If due to some by-product of metabolism there would also be considerable chance that if the animal lived a few days recovery would follow.

As already mentioned, I am of the opinion that the toxin does its work on the selected part of the nervous system, and puts it out of functioning for a considerable time. It does not appear to be a case of depressing or stimulating the different parts, but really it seems to be a matter of more or less destroying the nerve cells in the part affected.

If we watch a subacute case which recovers, we can hardly avoid coming to the conclusion that regeneration of nerve cells has had to take place before the normal functions have been able to return.

That the sympathetic is not the part destroyed is quite evident, but I am inclined to think the excessive sympathetic action is due to the depressed functioning of the parasympathetic due to more or less disablement of its peripheral cells.

Dickson and Shevky arrived at the conclusion that the toxin of the *Bacillus botulinus* attacked the peripheral nerve cells. Professor Greig's views on sympathicotonia would lead us to understand that he does not agree that the peripheral cells are affected in "grass disease."

I must say I have much sympathy with this view of the action of the toxin as expressed by Dickson, and think a somewhat similar disturbance occurs in "grass sickness."

Therapeutics. Glancing briefly at the therapeutics of the disease, the outstanding fact is that we have found no remedy of any marked value. It was at first thought that if we could induce purgation the patient would have a better chance of recovery, but we find that although we do get some cases on the border line between the acute and subacute which respond to active purgation, it merely converts what might have been an acute case into a subacute.

After carrying out some tests, I am quite satisfied that in an acute case there is neither absorption from the stomach nor any escape of contents through the pylorus

and that the oral administration of physic in these cases is a waste of time and money.

We are left with the intravenous and subcutaneous methods of injecting active remedies, and even by these paths we cannot say that any remedy of value has been found. In the subacute and chronic types of case any little improvement has followed the use of drugs which stimulated the parasympathetic. Any action produced by arecoline, physostigmine and pilocarpine is very temporary, and we would no doubt begin to note a more rapid improvement in these cases if the reaction could be prolonged for a time; but a mere half-hour's response followed by a return of the symptoms suggests that these remedies have not much practical value.

If the therapeutics are not promising, is there any prospect of a prophylactic?

This can only be answered when the cause is determined. Very few animals recover from "grass sickness," but among the few we have some which developed second attacks the following year. If a natural attack does not confer immunity it will be as well not to expect too much from artificial immunity. Perhaps Professor Pool will tell us whether, if the cause of grass disease should ultimately prove to be an ingested toxin, there would be much probability of a reliable prophylactic being prepared against it?

When research has so far failed to clear up the problem, can we suggest any variations in the farm management of the horses which might diminish the risk?

The best suggestion would be to keep inside in cattle courts and feed hay with a little oats. I also think that if horses are put more gradually to grass, being brought in at night for the first month, and stable fed, so that they enjoy a mixed diet, the risk, while not wiped out, is much reduced.

It is found in practice that these recommendations interfere with the established customs of the farm, and most farmers prefer to pay £5 per cent. to an insurance company to take the risk. The disappointing experience of past recommendations provokes sympathy with the writer of these lines:—

> I used to think I knew, I knew;
> But now I must confess:
> The more I know, I know, I know,
> I know, I know the less.

Mr. T. M. INGLIS (Forfar): You will expect me to say something in connection with this mysterious disease. I do not wish to attempt to criticise the papers, I only wish to say a few words as a practitioner. I am one of these unfortunates who is forced to reside in a district where the disease makes it seasonal visitations.

I do not agree with those who consider the disease a new one, having had similar cases of the acute type as far back as thirty years ago. I can remember on one farm three young horses dying of disease after exhibiting symptoms similar to acute "grass disease." The *post-mortem* appearances were difficult to understand and not what one would expect to find.

I do not recollect what opinion I expressed to my client, but he evidently was more satisfied than I was. The same season I had two similar cases on the neighbouring farm in two-year-old Clydesdales, and I can remember several single cases of the acute type from then on, but I cannot recollect a single case of the subacute type. In my experience the disease usually occurred on highly cultivated farms, but now it is found all over.

I have been associated with all the investigations from first to last, and am of opinion that too little attention has been paid in the past to the botanical side. I hold that the disease is one affecting the grass-eating animal; I would go further—I should say it is one of the grazing animal. I have never had a case in my long experience in an animal eating cut grass in the stable.

Horses when grazing snort a great deal, and in disturbing the dust it is possible that the infection may be brought about by inhalation.

Climatic conditions appear to control the disease to a great extent because during a spell of dry weather in June, for instance, we usually have a great many cases, then, if rain comes, the cases cease.

Cases of the disease do occur during the winter months, but usually on a farm. The farmers do not keep their horses in the stable; they have to work, and whether in field or stack yard the animals always have an opportunity of getting access to grass of some kind, which, in my opinion, explains these winter cases.

I have been particularly interested in Professor Greig's paper and his explanations of the clinical symptoms of the disease. To my mind, at least, these interpretations help greatly to lift the hitherto mysterious curtain.

The research worker has many obstacles to overcome, and the man in the street expects too much of him. He cannot rush to conclusions without exploring every possible source, and this requires time and great patience. I would like the Press here to give a hint to those interested that research requires financial support, and it is up to them to give that assistance. The research workers can rest assured that the practitioners will give them every assistance, as they have done in the past, and that they will always have access to their patients suffering from this troublesome disease.

Professor A. J. CLARKE, of the Chair of Materia Medica in the University of Edinburgh, said there was a strong possibility that the disease was caused by a toxin of some kind, and there was an extraordinary resemblance between it and over-excitation of the sympathetic system. The elucidation of the problem might be assisted if they knew more in detail of the elementary physiology of the horse. To his mind, the greatest need in the immediate present was for the practitioners and others to get the symptomatology more "cut and dried."

Dr. H. DRYERRE, Professor of Physiology, Royal (Dick) Veterinary College, Edinburgh, said he agreed that the bulk of the evidence suggested that the cause of the symptoms was a toxin. There did not seem as yet enough support for the idea that the toxin was introduced by insect bites. One was inclined to the view, therefore, that the toxin gained entrance by the alimentary tract. By this channel nature had erected two barriers to protect the animal; these were, the intestinal mucosa and the liver. If the former were damaged and a mass absorption of toxin occurred, the liver would be unable to cope with it, and this would precipitate an acute attack. Slight absorption of the toxin could be more easily combated by the liver. He suggested as a hypothesis that there might be two factors involved in contracting "grass sickness": a toxin, *per se*, and some irritant which so lowered the vitality of the mucosa as to allow the toxin to pass through. The irritant might even be sand or grit adherent to the grass, or stirred up from dry sandy soil by the snorting of the horse. This would be compatible with the observation that attacks of the disease ceased abruptly on the occurrence of rain. *Post-mortem* findings in the alimentary tract were not inconsistent with this hypothesis, investigators having reported the appearance of petechial hæmorrhages in the mucosa, and the accumulation of very hard fæcal masses. He suggested that the bowel stasis might be a precursor, rather than a symptom, of the disease. The toxin concerned might conceivably be derived from the partial hydrolysis of the proteins in the horse's diet.

In a letter which the Secretary submitted, Mr. HEWITSON, (Brampton, Cumberland), referred to the influence of weather on the disease. His experience was that the disease occurred mostly when the wind was in the east, and he knew that when the wind changed into the south-south-west cases ceased to occur. He thought that young immature, well-bred horses were more liable to be attacked by the malady.

Mr. JOHN BROWN, Invergordon, said he had seen 600 cases in the last ten years, and confessed that he knew as much about the disease now as he did when he saw the first case. It was a most mysterious disease. The

majority of the cases he had had were in single horses on farms where several horses were kept. Moreover, he had noticed that when a suckling mare was affected her foal was never ill. A peculiar thing in 1925 was that out of seventy-eight cases, seventy-four were of single horses on seventy-four different farms, pairs being affected on only two farms. During the last three years the disease had recurred on three or four farms where it had been before, but all the others were on new places, where it had not previously been seen. Another curious instance was that three horses died in one year on a farm where they had been grazing for five or six years. His experience was that the start of the disease in a season coincided with hot weather, and that it stopped if they got a cold wet week. He had had cases from April to October, but this year he had three in April, twenty-four in May, twenty-two in June, five in July, and since then there had been none. He was rather inclined to support the view of Mr. Inglis that the disease was caused by inhalation.

Dr. M. Wilson, Reader in Mycology and Bacteriology, Edinburgh University, speaking from the point of view of a botanist, suggested the query—Is the disease due to a fungus? He could see nothing to negative that possibility but at the same time he could see no definite evidence for it. It might be that some new fungi had been introduced into the herbage. The weather conditions under which "grass disease" appeared were those necessary for the spread of parasitic fungi, but the low incidence of the disease was certainly difficult to explain. They knew that the distribution of parasitic fungi was very erratic and they could not expect to find fungi present in a particular field or district every year. He thought there should be a more systematic investigation of pastures in which the disease had occurred. Although investigations had been made into the distribution of the flowering plants in the pastures blamed for causing "grass sickness," he believed that little or no work had been done from the fungus point of view.

Mr. A. Spreull, Dundee, said the question of the age at which horses were affected depended very much on the district. In his own district they had a great deal of arable land, and they had a preponderance of horses about five or six years of age. But he had had cases from yearlings up to horses whose age no man could tell. Regarding horses fed on cut grass becoming affected, he said he had had two distinct cases of "grass disease" among horses belonging to a cow feeder, and the grass fed to them was brought from a farm on which no "grass disease" had ever been, and he had attended that farm for the last fifteen years. This year he had one peculiar case where there was a lack of co-ordination in the movement of the horse. It was a subacute case and the horse eventually recovered. This year, whether due to the wet weather or not, they had certainly had far fewer cases than formerly. Speaking of the impaction of the bowel that occurs in affected horses, Mr. Spreull said that three years ago he was given the opportunity on a big farm of making careful daily observations of a stable of horses. They had four cases of "grass disease," and he was satisfied from the observations and examinations he made that impaction of the bowel of the affected animals occurred within a very short period, probably within twenty-four hours. Up to that time it was generally supposed that the cementation was a gradual process.

Mr. William Brown, Aberdeen, urged the importance of team work in carrying out investigations regarding grass sickness. He stressed the need there was for money to make research possible, remarking that if the farming world contributed a penny per week on their rent it would be one of the greatest insurances they could get. What had exercised his mind for years was—where did the disease come from? He did not agree with Mr. Inglis, but felt very confident that it was a new disease. He drew attention to the fact that the great majority of cases occurred on cultivated land, in fact in that respect they could almost draw a line between cultivated land and permanent pasture. He recalled that formerly no grass seed was imported, but they were now getting seed from Continental countries, from Canada, U.S.A., the Argentine, and other parts, and it might be, he thought, that with those importations of grass seed they had got the disease. Most probably the disease existed in those countries from which they got grass seed. If the disease was in the nature of a soil organism it would be washed back from the grass into the soil in wet weather.

Mr. John Brown, Invergordon, said he had seen many cases of the disease occur on permanent pasture and on a sandy moor near Tain.

Mr. Hugh M'Vean, Craigellachie, also said that he had seen cases on permanent pasture which had never received an ounce of seed or a pound of manure.

Mr. Nairn, Blairgowrie, reverting to Dr. Dryerre's suggestion, said the disease was localised, and the extraordinary thing was that it did not appear in other parts of the country where there was plenty of sand.

Mr. Dalling, London, of Messrs. Burroughs Wellcome and Co., remarked that if it was a toxic disease, it might not be due entirely to one toxin, and he was inclined to agree with Dr. Dryerre's idea of sand irritation.

Dr. Dryerre remarked incidentally, amidst laughter, that he had suggested a toxin *plus* an irritation caused by sand, and immediately a gentleman from Invergordon had blurted out that he had frequently secured cases of "grass disease" on sand hills in the north.

Professor W. M. Mitchell asked whether any record of surgical emptying of the cæcum through the flank existed. He suggested that it might be worth while attempting such an operation, possibly approaching this organ at its base, where there was no peritoneal covering. He pointed out that so far no medicinal treatment had been successful, so that surgical interference could not be less of a success.

Mr. Hugh Begg, County Veterinary Inspector for Lanarkshire, was rather intrigued by the theory put forward by Dr. Dryerre. He suggested that horses might be brought more gradually on to grass.

Mr. Wm. Robb, Glasgow, said Gamaliel and Co. had been very cautious in their pronouncements that afternoon. He had never had anything resembling "grass disease" in a city horse.

Mr. A. R. C. Bowie instanced two cases in which recovery appeared to follow injection of pituitrin. He would be glad if Professor Greig would inform him as to the action of pituitrin on peristalsis.

Mr. H. A. Watson said that since the adrenal medulla responded to painful stimuli such as occurred during an attack of colic, could not the sympathicotonia of "grass disease" be ascribed to the colicky symptoms with which the attack was often ushered in?

The Chairman, in closing the discussion, asked why it was they did not get the disease in the south of Scotland? "We put in as good seeds in the south as in Aberdeenshire. We have sand also, but I have never met a case of 'grass disease.'"

Mr. Wm. Brown, Aberdeen: You mostly graze your horses in the south on permanent pasture? (Laughter.)

The Chairman: Yes, when the grass seeds are a failure. (Laughter.)

The Replies.

Professor Pool: Most of the questions asked appear to have been answered by other speakers. The general discussion shows quite clearly that there is very little more to be learned regarding the etiology of "grass disease" from mere clinical observation, and that until more experimental results are obtained we are practically at a deadlock. Mr. Nairn asks if there is an increase of temperature in the early stages of the disease. It is impossible to answer this question at present. If a staff can be obtained for about six weeks from the middle of May and 1,000 farm horses in the "risk" area are observed to the extent of a daily observation of the pulse, respiration, temperature, state of the fæces and any other notable symptom, valuable information would be acquired because some of those horses would develop the disease, and in these cases the antecedents would be known.

Mr. Nairn doubts the possibility of the carcases of small animals lying on pastures and so providing a source of development of *B. botulinus* toxin. There appear to be relatively frequent opportunities for the bodies of small mammals and birds to lie about on pastures. They might not always be very apparent, as they might be hidden in holes or crevices. The botulism theory, however, appears to break down on the grounds that the "grass disease" syndrome differs from the botulism syndrome.

Mr. Nairn asks if there is likelihood of a successful method of prophylaxis, it the cause be an ingested toxin. Until the nature of the toxin be discovered (if such an agency is the cause of the disease), it will not be possible to answer this question.

Professor Dryerre advances a most interesting theory. I presume he uses the word "irritant" in a very wide sense and only suggests sand as one of the possible types of "irritant" capable of increasing the permeability of the intestinal mucosa to toxins. Horses, of course, thrive in sandy desert country and, although the ingestion of sand leads to attacks of sand colic, it does not lead to the development of the "grass disease" syndrome.

It will be interesting to find if other observers can confirm Mr. Brown's observation that foals at foot never take the disease. The fact that the diet of a foal at foot is chiefly or entirely the dam's milk, may of course be an important factor in this connection.

Dr. Wilson's remarks on the possibility of a parasitic fungus as the cause of the disease are most interesting. Although numerous observers have made extended search for such a fungus the results have been very unsatisfactory, and no one has isolated a fungus capable of causing the "grass disease" syndrome. It is, of course, possible that more extended search would give different results.

Mr. Spreull's observation that in two cases impaction occurred within an interval of twenty-four hours is invaluable, and I am glad to see this definite clinical observation placed on record.

There may be a great deal either directly or indirectly in the importance of imported grass seeds, as emphasised by Mr. W. Brown. It is a fact, of course, that in the course of the last twenty years the method of treating rotation pastures has undergone revolution and this interval coincides with the rise into prominence of "grass disease."

It is hoped that a detailed survey may be made in the course of the ensuing winter into the incidence of "grass disease" and its history during the last fifteen years. The correlation of the data obtained in the course of this survey may throw light on the importance of various factors in the spread of the disease.

Experimental and observational work will be carried out on pastures in typical parts of the affected area by the various specialists interested in the disease, and it is hoped that the sum of the work carried out in their own individual laboratories and in that of the Animal Diseases Research Association will throw light on the cause of "grass disease."

Professor RUSSELL GREIG: I am interested and rather gratified that none of the speakers has taken exception to the symptoms I have detailed as those of acute grass disease, nor to the parallel I have drawn between them and those of sympathicotonia.

It has been pointed out that the symptoms of sympathicotonia could be induced by any painful stimulation. While this is partly true, such effects are of a very transient nature and disappear upon the cessation of the pain. This is not comparable with the sympathicotonia of "grass disease," which persists for days and is even shown in cases of the sub-acute type.

Mr. Bowie instanced two cases which had responded to the injection of putuitrin. Pituitrin has been regarded as a direct stimulant to intestinal muscle, but recently, as the result of experiments upon small animals, this action has been doubted, and the effects of the drug upon peristalsis have been attributed to the histamine which is contained in its preparations. One cannot, however, argue from one species to another, and it is possible that pituitrin may be a direct stimulant to the equine gut.

I am certainly of opinion that, in those cases which are capable of such response, the rousing and maintenance of peristalsis constitute very important remedial measures.

Mr. Nairn accepted my parallel, but considers that the symptoms more probably represent a relative rather than an absolute sympathicotonia. My mind is still open upon the matter but, in considering the whole evidence, it seems to me that a positive sympathicotonia is the condition with which we are dealing.

It should be remembered that, while a lesion may occasion a failure in function, a so-called "discharging lesion" may exaggerate function, and to such lesions we could ascribe the sympathicotonia of grass disease.

But I cannot venture any firm opinion as to the condition which produces the sympathicotonia. My object to-day is to point out the striking and remarkable comparison which exists between over-activity (relative or absolute) of the sympathetics and the symptoms of acute "grass disease."

Further I do not propose to go.

I would take this opportunity of appealing for a broader basis for veterinary research.

The words "veterinary research" have come to be regarded as almost synonymous with bacterial and pathological investigation. Great as is the value of these sciences to veterinary medicine, only those who are in a position to appreciate the present lopsidedness in animal disease investigation, realise the need for a broader application of the biological sciences in the co-option of the comparative physiologist, the pharmacologist and the bio-chemist.

It has been said that, since the war, the nation cannot afford research, The truth is that agriculture cannot, now, afford to be without research, and the inclusion of these three workers would, in my considered opinion, give a powerful impetus to the progress of veterinary science.

The Scottish Agricultural Press is strongly represented at this meeting. Before concluding, I would refer to a matter directly concerning the agriculturist, but I shall try to do so as delicately as may be.

It has been the experience of veterinary research workers to be faced with lay criticism to the effect that results were obtained only very slowly. We, here, fully understand that this is in the very nature of sound research work, but to a large number of laymen, veterinary research appears to represent certain obscure and delicate manipulations conducted in a laboratory, which lead to the dramatic discovery of the cause and cure of the disease, within a period of a few weeks or at most a few months.

My reason for referring to the matter is that the pressure of lay criticism may be such as to tempt the researcher to be hurried into accepting incomplete and ill-considered evidence, and to reach definite conclusions which are later found to be erroneous and misleading.

Lay opinion should be informed upon the nature of research work and the time and labour required for its proper performance.

Savory and Moore, Ltd., chemists, of New Bond Street, W., have completed negotiations for the acquisition of John Bell and Croyden, Ltd., analytical dispensing chemists, of Wigmore Street, W.

Savory and Moore, Ltd., have been in New Bond Street for 150 years, and the firm of John Bell and Croyden is claimed to be one of the largest businesses of its kind in Great Britain.

The combine will also include the business of Arnold and Sons, surgical and veterinary instrument manufacturers, of Wigmore Street.

Readers may now secure, post free on application, from The Holborn Surgical Instrument Co., of 26 Thavies Inn, Holborn Circus, E.C.1, copies of the 1928 edition of their Veterinary Instrument Catalogue.

REVIEW.

[A Treatise on the One-humped Camel in Health and Disease. By A. S. LEESE, M.R.C.V.S.].

This book supplies a long felt want to veterinary surgeons and others who have charge of camels. Considering the great importance of this animal both from a military and civil point of view, no veterinary surgeon working in the East can afford to be without this treatise.

The mortality of camels during times of peace has been considerable and during the great war was very heavy; this mortality was greatly due to the fact that only very few veterinary surgeons had any experience or knowledge of these animals in health and disease—and there was no book with any detailed information available to which veterinary surgeons could refer.

In reading this treatise, one realised the amount of time and labour the author must have spent upon it—from his great experience of the camel in different parts of the world and extending over many years, his knowledge regarding the management of the camel in health and disease is necessarily very great; by publishing the result of his work amongst these animals he has filled a long felt want in veterinary literature.

The book is undoubtedly a complete practical guide to camel matters and veterinary surgeons working with these animals.

Not only the veterinarian but also the medical officer working in the tropics will find much of interest in the sections dealing with trypanosomiasis, etc.

Those sections dealing with the general management and the purchase and examination for soundness will prove invaluable to officers in charge of camel corps and to camel purchasing officers.

The various diseases and their treatment are fully dealt with and the sections dealing with them will be of the greatest help for veterinary surgeons serving in the East.

The author holds the view that camels are not susceptible to rinderpest—other workers in the tropics, with a considerable knowledge of camels and of rinderpest, maintain that the camel is susceptible.

A glossary of vernacular and technical terms is given at the end of the book and should prove of value.

This readable book is well printed—though the type is small—and is well illustrated. Both author and publisher are to be congratulated on issuing a most valuable treatise.

H.E.C.

H.M. STATIONERY OFFICE PUBLICATIONS.

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DIVISIONAL REPORT.

Dumfries and Galloway Division.*

ELECTION OF OFFICERS.

A meeting of the above Division of the N.V.M.A. was held in Dumfries on December 22nd, 1927. The following members were present: J. G. Bell, President; Messrs. Edgar, Lindsay, McAlister, Waddell, Milligan, Davidson, Simpson, Armstrong, Major Robb and A. Young (Hon. Secretary).

Surgical Demonstration.—Preceding the luncheon, a surgical demonstration was given in the Royal Auction Mart, Major A. ROBB (Glasgow) being the demonstrator. The operation was tenotomy of the peroneus tendon for the relief of stringhalt. Two subjects were provided and in each case the operation was performed in the standing position and a local anæsthetic used. The Major demonstrated in his usual facile manner and was followed closely by the company with keen interest.

After luncheon in the Station Hotel the business meeting was held. Apologies for absence were read from the following members: Messrs. Croudace, McKinlay, Davidson, Robinson and Scott.

New Members.—Messrs. Ferguson (Lockerbie) and Brennan (Penrith), proposed at the last meeting, were elected to membership of the Division.

ELECTION OF OFFICERS.

President.—The PRESIDENT (J. G. BELL) proposed, as his successor in the Chair, Mr. R. C. Robinson (Carlisle). This was seconded by Mr. MCALISTER and carried unanimously.

Vice-President.—Mr. MILLIGAN proposed as Vice-President Mr. R. Simpson (Carlisle), Mr. DAVIDSON seconded and this was unanimously agreed.

Auditor.—Mr. J. Lindsay was re-elected auditor.

Hon. Secretary and Treasurer.—Mr. A. Young.

N. V.M. A. Council Representative.—On the proposal of the PRESIDENT, seconded by Mr. MILLIGAN, Mr. Robert Simpson, Chief Veterinary Inspector, County of Cumberland, was appointed as the representative of this Division on the Council of the N.V.M.A.

MOTION BY MR. MILLIGAN.

Mr. MILLIGAN having given notice, moved that the N.V.M.A. should be asked to ascertain the legal aspect of a circular letter, sent by the Dumfries County Council to veterinary surgeons practising in the county, in which it is stated that the fee of 2s. 6d. will not be paid for notifications under the Animals (Notification of Diseases) Order of 1919, or the Tuberculosis Order of 1925, unless such notifications were made on the forms prescribed by the local authority.

This was seconded by Mr. LINDSAY and very fully discussed, all members taking part in the discussion.

Correspondence.—(1) From the N.V.M.A., with reference to the formation of a Veterinary Inspectors' Section within the Division. After discussion, it was decided to take no action.

* Received on 4th January.

(2) From Dr. Leslie Sheather, asking the Division to support his application to the Home Office for registration of his private laboratory lnder the Vivisection Act. The Hon. Secretary, after consulting the President, had replied giving their support. This action the meeting approved.

Nomination to Membership.—Mr. Sangster (Dumfries) was nominated as a member.

Next Meeting.—It was decided, after discussion, that the next meeting of the Division be held in the latter part of June and that it take the form of a social gathering of members and friends, at some place of interest to be decided by the President.

A. YOUNG, *Hon. Secretary.*

IRELAND AND FOOT-AND-MOUTH DISEASE.

"For several years past there have been periodical outbreaks of foot-and-mouth disease in England, and just now it has appeared in seven shires. Irish farmers will sympathise with their English confrères in the losses and inconveniences entailed by the restrictions deemed necessary to stamp out the disease," says *The Cork Examiner* of 5th January. "But Irish farmers on both sides of the boundary will join in protesting against irresponsible and uninformed statements made by members of English public bodies who try to place the blame on Irish cattle. At a meeting of the Hereford Agricultural Committee one Alderman Hall declared that the whole trouble was due to disease in cattle leaving Ireland. He knew from experience that Irish cattle were full of disease, and all reports to the contrary were untrue. Evidently the other members of the Committee were not of the same opinion, and one blamed Argentine cattle, but Mr. Hall persisted that Ireland was the only place to blame. He supplied the information that Argentine cattle were rigidly inspected before shipment. Apparently Mr. Hall, for all his experience and all his knowledge, is unaware that all cattle leaving Ireland are examined by veterinary surgeons for symptoms of foot-and-mouth disease, and are detained long enough at the ports on both sides of the Channel for the disease to develop into the acute stage, before being allowed to proceed to the English destination. No case of foot-and-mouth infection has been traced to Ireland for a number of years. If Alderman Hall of Hereford thinks otherwise he should place his case before the authorities, who could then sift his evidence. Farmers and veterinary surgeons of Northern Ireland, as well as the Free State, will repudiate Mr. Hall's uncorroborated statements."

. . . . . .

"The statement made by Alderman Hall at the Hereford Agricultural Committee regarding Irish cattle was officially denied on inquiry at the Northern Ireland Ministry of Agriculture to-day," says the *Irish Times* of the same date. "The statement was characterised as ridiculous. There has not been an outbreak of foot-and-mouth disease in Ireland since 1921.

"It was also pointed out that all cattle are inspected before leaving Ireland, and upon their arrival in England are detained for a period of six days under existing regulations, and are again subjected to inspection. There is first a ten hours' detention at the port of disembarkation, and inspection by the port authority, and for six further days they are under observation before being exposed for sale in any market."

Cattle plague has been prevalent, with varying intensity, in Palestine, Transjordan, the Lebanon, and Syria since the summer of 1926. On 15th January the declarations were published which have been exchanged between the High Commissioners of Palestine and Syria, announcing that these areas were free of plague and also the removal of restrictions.

NOTES AND NEWS.

The Editor will be glad to receive items of professional interest for inclusion in these columns.

Diary of Events.

Jan. 23rd—Finance Committee N.V.M.A., 4 p.m.
Meeting of the Editorial Committee, N.V.M.A., 4-30 p.m.
Propaganda Sub-Committee of Organising Committee at conclusion of Editorial Committee.

Jan. 25th—Meeting of the Scottish Metropolitan Division, at Edinburgh.

R.C.V.S. COUNCIL ELECTION, 1928.

The nominations given below have been made to date for the forthcoming election to Council R.C.V.S. Names marked with an asterisk are those of serving members who are nominated for re-election. There are eight vacancies.

Name.	*Address.*	*Proposers.*
*Buxton, James Basil	Cambridge	O. Charnock Bradley. Sir John M'Fadyean.
*Clarkson, J. ...	Garforth, near Leeds	O. Charnock Bradley. J. Abson.
Downham, Kenneth Douglas	Liverpool ...	J. H. Wright. H. Sumner.
*Locke, G. H. ...	Manchester	J. W. Brittlebank. J. H. Wright.
*M'Fadyean, John ...	Leatherhead, Surrey	O. Charnock Bradley. J. W. Brittlebank.
Martin, G. Dunlop	Westcliff-on-Sea	F. T. G. Hobday.
Rabagliati, Duncan Silvestro	Wakefield, Yorks.	J. Clarkson. J. Abson.
*Rees, W. D. ...	Penygraig, Glam.	R. Moore. R. W. Hall.
*Share-Jones, J. ...	Liverpool ...	J. M'Fadyean. J. W. Brittlebank.
Sheather, A. Leslie...	London ...	J. W. Brittlebank. J. M'Fadyean.
Walker, G. K. ...	Abingdon, Berks.	F. T. G. Hobday. J. Moore.
*Willett, John ...	London ...	J. W. Brittlebank. O. Charnock Bradley.
*Wooldridge, Geo. Henry	London ...	J. W. Brittlebank. O. Charnock Bradley.

R.C.V.S. OBITUARY.

PLOMLEY, William, M.R.C.V.S., Melrose, Tankerton-on-Sea, Kent. Highland and Agricultural Society, 1875. Graduated, Edinburgh, 9th August, 1879. Died, 10th January, 1928, aged 76.

. . . . . .

WALLIS, William Dawson, M.R.C.V.S., 74 Wood Street, Barnet. Graduated, Edinburgh, May, 1895. Died, 10th January, 1928.

. . . . . .

SANDERSON, Fred Hall, Victoria Road, Darlington, Co. Durham. Graduated Edinburgh, 18th December, 1909. Died, 12th January, 1928, aged 43 years.

"Mr. Sanderson had been at Darlington since 1912, when he took over the practice of the late Mr. Furness, and had built up a very extensive and successful business," says the *Newcastle Daily Journal* of 4th January. "He had charge of the Marquess of Londonderry's stud at Wynyard, and also the stud of the Zetland Hunt. He had been veterinary inspector to Darlington Corporation since 1920.

"Mr. Sanderson was the son of a well-known retired Northumberland farmer. After passing his examination as a veterinary surgeon, he was employed as assistant veterinary surgeon by the Great Western Railway Company in London from 1910 to 1912. He was also for some time with the late Mr. E. H. Leach at Newmarket.

"During the war Mr. Sanderson served with the Army in India, attaining the rank of captain. A well-known Freemason, Mr. Sanderson held the rank of Junior Deacon in the Restoration Lodge (Darlington), and he was also a member of the Mark Lodge of Freemasons. He was also a popular member of the Darlington Quoit Club.

"His wife, who survives him, is a daughter of Mrs. Dods and the late Mr. W. P. M. Dods, of Denton Hall, and there are three young children."

The late W. A. Taylor, F.R.C.V.S.

An Appreciation.

The veterinary profession in Lancashire has suffered a further severe blow by the death of one of its most respected and beloved members in Mr. W. Augustus Taylor, F.R.C.V.S., who died on the 7th instant, as announced in last week's *Record*.

It is difficult to do justice in the narrow space of an obituary notice to the memory of one who played so conspicuous yet modest a share in the progress of his beloved profession during what may be described as probably the richest period of its professional progress.

Qualifying from the Royal Veterinary College, London, in 1870, Taylor returned to assist his father in the old-established practice, and when Peter Taylor died it fell to his lot to carry on the great responsibilities attaching to his heritage. How great these obligations and responsibilities were is only known to those who were privileged to enjoy his close friendship and intimacy, but they were a heavy burden for a young man.

"Gus," however, as he was ever known to all of us, fulfilled his trust in the way that he did everything in his life. His difficulties were great, but to him they were obstacles to be overcome.

His professional reputation grew rapidly, and it is no exaggeration to say that there was no veterinary surgeon in Lancashire whose opinion was more valued throughout these years; and Lancashire, during the period referred to, was singularly rich in veterinarians of more than ordinary capacity. One has only to refer to the names of the late Sam Locke, Alexander Lawson, Edwin Faulkner, Tedbar Hopkin, John Wolstenholme, all of whom were contemporaries of his, to realise what a great group were these Manchester men during these years.

As a consultant his services were in great request, and many a young practitioner can look back to the kindly advice and encouragement that was ever available when meeting him to settle some knotty technical question of diagnosis or treatment.

Despite the responsibilities of a wide and ever-growing practice, Mr. Taylor was indefatigable in all questions affecting the welfare of the profession, and although he never served on the Council of the Royal College of Veterinary Surgeons, had a great influence in professional politics generally, and his advice and opinions were eagerly sought by those whose connections in these matters were more intimate.

He was appointed to the Examining Board of the Royal College of Veterinary Surgeons in 1893 and was twice President of the Lancashire Veterinary Medical Association, whose members only four years ago elected him an honorary member in memory of his devoted services to the Association, and at the same time presented him with a service of plate to record the affection and esteem in which they held him.

He was Honorary Veterinary Surgeon for very many years to the Altrincham Agricultural Society, and intimately associated until his death with the Manchester Dog Show. He was also for a time President of the National Master Farriers' Association of Great Britain.

Apart from all this, however, and if he had done nothing else, his great work for benevolence would have earned our everlasting gratitude. A member of the National Veterinary Benevolent and Mutual Defence Society since his entry to the profession, Vice-President from 1896-1902, then continuously President until 1927, his life's desire was satisfied, as he saw the Society from year to year growing in strength and capacity for good until it has become an integral part of professional life.

Straightforward and upright as he was himself in all his dealings, and believing really that every able-bodied man should carry his own burdens, he was ever charitable in his judgments and regarded the duty placed upon us to help the sick and needy, the widows and orphans, as a sacred obligation which was not lightly to be set aside.

In conclusion I may perhaps quote an expression by one who had just heard of his passing which perhaps expresses our feelings most adequately—"Dear old Gus, we shall miss him, but his memory will not fade."

. . . . . J. W. B.

The deceased gentleman was interred at St. Luke's, Cheetham Hill, Manchester, on Tuesday, January 10th, the preceding service being held at St. Mark's Church. Amongst the large company present were the following members of the profession: Mr. G. H. Locke (representing the R.C.V.S.), Mr. A. W. Noel Pillers (representing the N.V.M.A.), Messrs. J. B. Chadwick, E. H. Curbishley, J. F. Dixon, J. W. Ingram and J. H. Wright (members of the Lancashire Division, N.V.M.A.), Mr. R. H. Oddy (assistant to the late Mr. Taylor), Mr. R. C. Locke (representing Lt.-Col. J. W. Brittlebank), Mr. F. Hopkin (Treasurer, National Veterinary Benevolent and Mutual Defence Society), and Mr. W. Woods (Vice-President, National Veterinary Benevolent and Mutual Defence Society). There were also present many persons prominently connected with various kennel organisations, with which Mr. Taylor was closely identified.

Personal.

Appointment.—Mr. S. J. Edwards, B.Sc., M.R.C.V.S., has joined the staff of the Institute of Animal Pathology, Camden Town, as Junior Research Assistant.

"Fright Disease" among Dogs.

London College to undertake Research Work.

"The increasing prevalence of what is known as canine hysteria, or 'fright disease,' is causing anxiety among lovers of dogs in various parts of the country," says *The Times* of 17th January. "This disease, the symptoms of which are sudden terror, causing the dog to froth at the mouth, to howl, to turn in circles until exhausted, or to run wildly until arrested by some obstacle, such as a tree or a lamp-post, was first noticed in this country about a year ago; but it has been observed in America for several years.

"Gun dogs were at first affected in Great Britain, and now the disease has spread to various other breeds, including pet dogs. The symptoms mentioned, which appear to be similar in every case, last for only a few minutes. The dog so affected does not become ferocious, but there is a danger that any person attempting to handle the animal incautiously when it is suffering from the disease may be bitten.

"Research into the causes of the disease is to be undertaken by the Royal Veterinary College, Camden Town. Professor F. T. G. Hobday, the Principal, stated yesterday that the work of the college is handicapped by lack of funds, and that a sum of £20,000, to establish a research scholarship and secure the necessary equipment, is required to carry out this investigation. He added that cases of 'canine fright,' the name by which the disease was known in America, had been reported from many parts of England and Ireland. A veterinary surgeon should be sent for as soon as a dog showed signs of being affected by the disease and the animal kept indoors for two or three weeks and carefully watched."

Foot-and-Mouth Disease.

That foot-and-mouth disease was brought to this country from the Continent by motor cars was a suggestion put forward by Mr. Harold Jackson, chairman of the Central Council of Milk Recording Societies, speaking on Saturday at a farmers' dinner at Preston. The source of

the spreading of the disease in this way, Mr. Jackson said, was the mud picked up on Continental roads and dropped in this country from the floor boards of cars. Mr. Jackson quoted the experience of a veterinary surgeon in support of his contention.

Abolition of the Lance.

The War Office and an Historic Weapon.

It has been decided, states an Army Order, to abolish the lance as a weapon of war. Henceforth it will not be carried on field training, but will be retained by lancer regiments for ceremonial purposes only. Training in the handling and use of this weapon, other than for ceremonial purposes, will be discontinued forthwith.

Under this new Order one of the oldest weapons in military history disappears, for all practical purposes, from the British Service. The lance—in ancient days the spear—disappeared from European warfare for a space after the introduction of gun-powder and the consequent decline in military value of the armoured and mounted knight. It had temporary revivals, but Napoleon was the first military leader in modern history to restore the lance as a weapon of first importance in the battlefield. His lancer regiments acquired a deserved reputation in shock attack, and they were employed with a direct reference to particular tasks. Because of his successes with lance-armed cavalry, other countries—Germany and England particularly—produced numbers of lancer regiments. England, for instance, converted many light dragoon regiments—the 17th is a notable case—into lancer units. For a time British regiments had lance-carrying front ranks, but that practice died with the Great War.

The change is unimportant in itself, but it will be taken in many quarters as an implication that the "mechanization" school is gaining points, and that those who desire to see a great reduction in cavalry strengths are, perhaps temporarily, in the ascendant.—*The Times.*

Clydesdale Society and Sound Mares.

At a Council meeting of the Clydesdale Horse Society, Mr. William Brown, Aberdeen, was appointed as veterinary surgeon in the examination of animals competing for the Society's two Cawdor Cups next season.

The reference from last meeting for a conference with the Board of Agriculture of Scotland regarding the recommendations from northern horse-breeding clubs on the subject of unsoundness was reported upon. This conference was held on December 1st, and it was agreed to recommend to the Board of Agriculture that, in the administration of their scheme for the improvement of heavy horse breeding, grants should be paid only in respect of mares whose owners furnished, before service, a certificate that the mares are, to the best of their knowledge and belief, free from shivering, and that in those cases where such a certificate cannot be given examination by a veterinary surgeon should be insisted upon. It was also agreed that the Board should prepare a draft form of certificate which mare-owners should be requested to sign before a mare is served.

The Council of the Clydesdale Horse Society agreed to adopt this recommendation and agreed further to suggest to district horse-breeding societies that they take steps to discourage the serving of mares known to be affected with shivering.

The Yorkshire Agricultural Society have proposed that three classes for Clydesdales be included in their 1928 Show, to be held at Halifax: one for yearlings of both sexes, one for two-year-old fillies, and one for mares of three-years-old and upwards. If that classification is confirmed, the Clydesdale Horse Society will grant two silver medals and £10 towards the prize money.—*Glasgow Herald.*

Horses in the Show Ring.

Methods of Handling.

"A case which has recently been heard in the Law Courts emphasises the fact that horses are frequently handled in a very perfunctory manner at our horse shows," says a *Times* correspondent. "Nothing can be more absurd than for the man who is showing the horse to stare him full in the face, at the same time putting all his weight into the halter or leading rein. Naturally the horse hangs back and stops, or tries to stop; he has received the signal to do so. It may fairly be said that a class of horses is seldom led into the ring without a considerable proportion of them being shown badly. Even at the most important shows, if there is a good strong class in the ring, a considerable proportion of them will be found standing anyhow when the judges are looking elsewhere. This means that, owing to the gross carelessness of those in charge, there is considerable risk of bad accidents to man or horse, or both. There was a time when such a state of things could not have prevailed, when the man who took a horse into the ring was a competent, if not a finished, horseman, and when the ring steward would have seen to it that the horses were properly handled, or else turned out of the ring.

"It is high time that ring stewards were invested with the authority which their predecessors had, and that, if they do not exercise it, they should make way for those who will. Then, again, it should be seen that the exhibits stand in their proper place, and that they are properly described. If these matters are not attended to, those who evade them should be disqualified. If a few 'examples' were made we should soon have a more creditable state of things in our show yards."

CORRESPONDENCE.

Letters to the Editor should reach the Office not later than by the first post on Tuesday morning for insertion in following Saturday's issue.

All correspondence must bear the name and address of the contributor for publication.

The Editor does not hold himself responsible for the opinions of his correspondents.

Rebuilding of the Royal Veterinary College.

To the Editor of the Veterinary Record.

Sir,—Fellows of the Central Veterinary Society were last Thursday treated to a very eloquent address by Professor F. T. G. Hobday upon the sad state of ruin of the Camden Town College. The series of pictures thrown on the screen was in itself sufficient testimony to England's shameful neglect of veterinary science. In the discussion which ensued, nobody mentioned the question of how funds could best be collected and there was no effort to enlist the personal services of each individual present.

It occurs to me, however, that if each individual in the veterinary profession would show some practical sympathy, the desired £250,000 might very soon be found, for, as Professor Hobday said, it only needs 250,000 sportsmen out of our 48,000,000 population to subscribe £1 apiece, to achieve the desired result.

I have suggested to the secretary of the fund that if he would print single-leaf pamphlets of appeal, I am sure a great number of veterinary surgeons would each take a supply, and enclose one of them in every letter he sent to clients, friends or business people. In this manner the appeal would be afforded an exceedingly widespread publicity. I could personally very soon dispose of 500. It seems a scheme worthy of consideration.—Yours faithfully, Hamilton Kirk.

67 Wigmore Street, Portman Square, W.1.

16*th January*, 1928.

Tuberculosis Eradication.

To the Editor of the Veterinary Record.

Sir,—I am surprised that Mr. Henry Gray should say that the eradication of clinically-tuberculous animals is moonshine.

In a country like ours the Government cannot order things just as they choose. Public opinion must be formed before any great step can be taken.

Those of us who have been accustomed to cattle practice or have had experience of the *post-mortem* examinations of cattle know what an awful financial loss tuberculosis causes the agricultural community. We also thoroughly believe those of the medical profession who assert that it (bovine tuberculosis) causes untold suffering to our children. But do the general public ? I emphatically say that they do not. This being the case, how can a Government be expected to bring in legislation for the destruction, with compensation, of thousands of *apparently* healthy ?

The stock-owner would object to his *valuable* breeding stock being destroyed, the public to the expense in extra taxation and the price of milk during the exterminating process.

The Tuberculosis Order, which has for its aim the destruction of clinically-affected animals is or can be made a most valuable means of combating the disease if every advantage is to be taken of it, if those who have the administering of it will undertake it in a right spirit and use it as a means of educating stock-owners.

Through the Order we are called to examine and advise on a great number of animals which, but for the Order, we should never see. The owner looks on these as "wasters," and one of the inevitable trade losses. He has been accustomed to get rid of them as best he could and forgets that they existed.

The Tuberculosis Order now causes him to report such an animal ; it brings a veterinary surgeon to the animal ; it opens the door for a discussion on the subject of tuberlosis and, in so doing, it gives the veterinarian an opening such has he has not had before of explaining the disease to the owner ; it gives the opportunity for a practical demonstration at the *post-mortem*. I think when the Order gives these opportunities to the veterinary profession to use, if it will, in the creation of a desire in the minds of the stock-owners to get rid of the disease, it is scarcely to be dubbed moonshine.

Mr. Gray says that it would be a much easier task to free this country than the U.S.A. I fail to see why it should be easier. Is it not a fact that the more cattle are housed the more liable they are to contract the disease ? Therefore, the more they are kept in the open air the more easy should it be to adopt means to exterminate the disease.

In Great Britain most of the cattle are housed in winter. I imagine that the percentage of housed cattle in the U.S.A. would be a mere trifle compared with ours.

The Milk and Dairies Legislation is framed to prevent the use of milk for human consumption from clinically-affected tuberculous animals, also to provide better hygienic conditions in the houses in which cows are kept.

The first will give some protection to the public ; the second will greatly help to check the spread of tuberculosis, especially when the clinically-affected animals are eradicated from the houses.

Surely, Mr. Editor, if we grasp the opportunities which we have been given instead of grousing because we have not got everything, there is a chance of creating that public opinion which would make easy the application of still further legislation against this serious plague.

There is no doubt that a demand will spring up for tested cattle to take the place of those condemned and slaughtered. The farmers are crying out for help. There is no doubt that the Government would be pleased to find some real means of helping them. Is it beyond the power of the veterinary profession to point out that if they would *help* and encourage breeders and dairymen to test their herds, provided that they in return adopted reasonable measures for getting rid of the disease, they would be doing a real service to the stock-owner and dairyman, and to the public ?—Yours faithfully, E. H. Pratt, M.R.C.V.S.

Absence of Oestrum.

To the Editor of the Veterinary Record.

Sir,—It would be of great interest and assistance to me to have the views of other members of the profession on the following. In one of my client's herds there are eight cows which have been calved for several months and which, so far, have shown no signs of œstrum. I have treated them with all the better-known aphrodisiacs, have tried massage of the ovaries, and finally powders containing 15 grains of cantharides, but with no result. The cows are otherwise in normal health and milking well.

No roots or other green foods are being fed, the only thing in the diet to take the place of these being sugar beet pulp, which is given about 3 to 3½lbs. per cow daily.

Is the condition, then, due to the lack of vitamins in the food ? Last winter roots were fed until the middle of November and no trouble such as the above was experienced.

My client has now purchased a stock of roots and we are hoping that this may provide a way out of the difficulty.

I would be greatly obliged by having the views of others who may have had the same trouble.—Yours faithfully, R. Turnbull.

Radstock House, Lewes, Sussex.
January 10th, 1928.

"Grass Disease" Research.

To the Editor of the Veterinary Record.

Sir,—During the last 25 years I have from time to time come across cases showing all the symptoms of "grass disease," as described in to-day's issue and by Professor Wooldridge in the *Encyclopaedia*.

All these cases have been grazed on marshes within a very few miles of London. The cases have been much more frequent in hot, dry summers and have been attributed to all sorts of causes—amongst others, sunstroke, and to the myriads of mosquitoes which infest this locality.

The owners of horses say that if they remove the patients to upland pasture as soon as the first symptoms are observed they recover.

For obvious reasons I cannot make the name of the district public, but if any members of the profession are desirous of prosecuting research into the matter I shall be pleased to place any data I possess at their disposal and it should be easy to arrange for grazing an experimental subject on the land during next summer. Faithfully yours, David Eric Wilkinson.

4 The Terrace, Woodford Green, Essex,
14th January, 1928.

"Unqualified Men."

To the Editor of the Veterinary Record.

Sir,—Mr. Fenn-Smith's letter is timely, but I go further than he did and say that the Council should do something to protect us against the "quack." In this district there are two, who cut prices and cripple us at every turn and yet we have no redress. Nothing can be done against them unless they call themselves veterinary surgeons, which they are too careful to do. Would any profession other than ours be so longsuffering ? It is time the "National" or the R.C.V.S. Council did something to protect us against unqualified men.—John M. Buchanan, M.R.C.V.S.

Stowmarket, Suffolk.

The Editor acknowledges the receipt of the following :—Communications from Miss D. Knight (Reading), Lt.-Col. J. W. Rainey and Major-General Sir Frederick Smith.

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